CUG-H00635-4.1-P16264

BRAIN

A JOURNAL OF NEUROLOGY

MACMILLAN AND CO., LIMITED
LONDON - BOMBAY - CALCUTTA
MELBOURNE

THE MACMILLAN COMPANY
NEW YORK - BOSTON - CHICAGO
ATLANTA - SAN FRANCISCO

THE MACMILLAN CO. OF CANADA, LTD.
TORONTO

41

105-
.002

Brain

A Journal of Neurology

105.
.002

EDITED BY

HENRY HEAD M.D. F.R.S.

WITH THE HELP OF

. M. BAYLISS, F.R.S.
YROM BRAMWELL, M.D.
FARQUHAR BUZZARD, M.D.
DAVID FERRIER, M.D., F.R.S.

GORDON HOLMES, M.D.
SIR FREDK. MOTT, M.D., F.R.S.
R. PERCY SMITH, M.D.
C. S. SHERRINGTON, P.R.S.
JAMES TAYLOR, M.D.

VOLUME XLIII.

MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON
W YORK: THE MACMILLAN COMPANY
1920

[MAY, 1920.]

CALCUTTA UNIV
Sec. No.
Book No.
LIBRARY.

BRAIN.

PART I, VOL. 43.

THE RESULTS OF SECONDARY SUTURE OF PERIPHERAL NERVES.[1]

BY JOHN S. B. STOPFORD, M.D.
Professor of Anatomy, University of Manchester.

	PAGE
I.—INTRODUCTION	1
II.—ANALYSIS OF RESULTS	5
(*a*) General	5
(*b*) Explanation of Charts	6
(*c*) Musculo-spiral Nerve	7
(*d*) Median Nerve	13
(*e*) Ulnar Nerve	16
(*f*) Sciatic Nerve	20
(*g*) External Popliteal Nerve	20
(*h*) Internal Popliteal Nerve	23
III.—CONCLUSIONS	23

I.—INTRODUCTION.

ALTHOUGH further time must elapse before the end-results of secondary suture of peripheral nerves, injured in the war, can be judged in large numbers, it is now possible to communicate a preliminary report upon the progress to date of those patients in which this method of surgical treatment has been found necessary; and it seems advisable to do this for the benefit of the many pensioners who still await further treatment.

So many factors may influence the progress and extent of recovery that it is exceedingly difficult to interpret all the observations, or estimate the influence of any one individual factor. To attempt the elucidation of the numerous problems encountered, or to provide any guidance for the future, it is obviously necessary to consider large numbers of patients on whom regular systematic examinations have been made and careful records kept of all complications, methods of treatment, and progress. No trustworthy conclusions can be drawn from the publication of a few isolated cases, or even from a large series, unless attention has been directed to all the factors which may prevent or cause delay or acceleration of recovery. The closest co-operation between the surgeon and the investigator is essential; if the latter is unable to be present at the operation, it is of fundamental importance that the most detailed notes should be provided of the anatomical and pathological conditions found on exposure of the nerve, together with an exact account of the surgical procedure adopted. It has been my good fortune to have the fullest possible support from my surgical colleagues at the Grangethorpe Hospital, Manchester, and I would like to express my deep appreciation of the careful and elaborate system of note-taking which they have instituted, as, without the information supplied by them, it would have been impossible to attempt an inquiry into the problems of secondary suture.

---

[1] A Report to the Medical Research Council.

In this report are included all the results of secondary suture, where it has been possible to continue observations for a period of not less than six months after the date of the operation. It was rarely possible to follow the patients even for so short a time before 1917, when the special hospitals were instituted; consequently almost all the patients quoted have been operated upon since the beginning of that year, and only a small number can be considered as end-results.

Altogether 271 examples of end-to-end suture are under review, and the majority of the patients have been examined at regular intervals of from four to six weeks for varying periods up to three years after the date of the performance of the suture.

As a preliminary it is convenient to consider the following general factors which may influence the progress and result.

(1) *The interval which has elapsed between the reception of the injury and the date of the suture.*—It is necessary, in connexion with this, to take into account the cause of the delay. If the operation has been deferred because of the presence of sepsis, widespread intraneural changes are likely to be encountered which will interfere seriously with, or prevent, regeneration. Excluding such a condition, a delay of twelve to eighteen months appears to have no marked effect upon the date or extent of recovery. If the interval exceeds this time, in my experience, the prognosis is not so good when the suture has been performed in the distal part of the limb; whereas, in the proximal part, a delay of two or three years does not seem to prejudice the chances o success. Suture of the median in the upper arm has been successfu

in two patients where there was an interval of two and a half years. A delay of three and a half years (the longest encountered) occurred in two patients, with injury to the median at the elbow. In one the only muscle to recover voluntary power was the flexor sublimis digitorum; in the other patient it is too early yet to give an opinion, but the signs are not hopeful.

(2) *The surgical technique adopted.*—In about 80 per cent. the same procedure has been used; so that in this series this factor is almost constant, and, consequently, no opinion can be expressed upon the advantages of the various suture materials. After free exposure, the extremities have been excised until distinct nerve bundles could be seen, and then the ends brought into as perfect apposition as possible without distortion. Whenever practicable, only sheath sutures of catgut have been employed, but in the larger proportion one tension suture through the substance of the nerve was found necessary. On comparing the progress and results, with and without tension sutures, there is no doubt that the effect of the through-and-through stitch is perceptible.

For some time my surgical colleagues have used no protective material to surround the line of suture; a new bed for the nerve has been constructed, or its anatomical course modified, so that the injured segment of the nerve lies only in relation with uninjured muscles. The clinical records clearly indicate the advantages of this over any protective material, either autogenous or heterogenous. Several nerves have been re-exposed by my colleague, Mr. H. Platt, after various materials have been used, and it has been proved that neither fascia lata nor fat prevent adhesions, and in two patients, where Cargile membrane had been employed, there was a thick collar of fibrous tissue around the nerve which in one was undoubtedly causing considerable constriction.

The prognosis is bad when a satisfactory bed cannot be procured, and the nerve has to be left in relation with scar tissue, a condition unfortunately encountered frequently when the external popliteal nerve has to be sutured in the region of the head of the fibula.

Displacement of the ulnar nerve in front of the internal condyle of the humerus has not, as far as I can ascertain to date, retarded the progress.

It is somewhat surprising that the extensive freeing of the nerve necessary in this manœuvre, or in other nerves when a large defect has to be bridged, has not had a more profound effect. I have insufficient

CUTTA UNIVERS

records yet to give an opinion upon the results after the two-stage operation.

The after-treatment has been the same—constant support of the paralysed muscles, interrupted galvanic stimulation of the muscles and massage, and later faradic stimulation and muscle re-education.

(3) *The condition of the nerve found on exposure at operation.*—Those patients in whom a complete solution of continuity was discovered at operation have tended to make a more satisfactory recovery than those in whom incomplete division was found, but I am convinced that this difference is very much reduced, if not eliminated, when a liberal resection is performed. Interstitial changes are frequently very extensive after partial division, and from the histological examination of pieces of nerve excised at operation there appears to be a tendency not to resect sufficiently widely. Free resection is particularly necessary after prolonged suppuration. Contractures and other mechanical disabilities are associated commonly with incomplete division and may be responsible for imperfect functional recoveries, although good regeneration may have succeeded the suture of the nerve.

(4) *The effect of some of the more frequent complications.*—Allusion has been made to the fact that extensive and prolonged sepsis in the neighbourhood of the nerve may influence seriously the prognosis. Microscopic examination has shown that, under such a condition, intraneural changes may be discovered as distant as 8 in. above the point of injury.

Ligature of the main artery in the proximal part of the limb, as the upper part of the brachial or the axillary, tends to retard the progress and, after suture of the ulnar or median nerves, appears sometimes to reduce the extent of the recovery of function, but it is difficult to exclude the possibility of this being due to secondary changes in the muscles as a result of the ischæmia. Involvement of the main blood-vessels in the distal part of the limb, as may be anticipated, does not usually prejudice the result.

Ununited fractures of the humerus present a serious complication. In four patients exhibiting this complication suture of the musculo-spiral was performed before the ends of the bones had been fixed. Two were complete failures, the third showed merely recovery in the supinator longus and extensor carpi radialis longior, whilst the fourth progressed favourably until a bone graft operation was succeeded by a recrudescence of sepsis, which resulted in complete loss of conduction and failure of regeneration. These experiences seem to prove that and yet the hand is of slight practical service, when the patient attempts purposive movements. The patient will often volunteer the information that when he attempts to work he finds that he loses the grip of his tools, and on inquiry it will be found that he can use the hand fairly well as long as he concentrates upon the movements, but the hand ceases to function satisfactorily as soon as he takes his eye off it. Such patients also frequently inform you that the hand is useless in the dark, or when they cannot watch what they are trying to do with it. Such complaints may be heard even when cutaneous sensibility has made a fair recovery and all the muscles, tested individually, exhibit voluntary power. It seems clear that such a disability is due to the loss of afferent stimuli from joints, muscles, tendons and other deep structures. It is only practicable to investigate directly the recovery of fibres conveying conscious impressions from such deep structures as joints, but from an investigation of the sense of posture and the appreciation of passive movements in the finger and thumb articulations supplied by the nerve, it has been found that these were generally lost, or at the best very defective even when three years had elapsed since the time of the suture. It would seem that this is very important to remember at examinations for the assessment of pensions after nerve suture in the upper limb, since the routine investigations of voluntary power and cutaneous sensibility do not, in themselves, provide sufficient information to determine the real functional capacity of the hand. It is also an awkward problem to contend with during the later stages of recovery when muscle re-education becomes of greatest importance, since it provides one of the main obstacles to the satisfactory application of this form of treatment—an obstacle which is probably insufficiently appreciated. Previous to the war our chief experience of muscle training was derived from the treatment of infantile paralysis, and consequently many who are responsible for the supervision of this form of treatment are content merely to develop an increased range of movement of the individual muscles, and fail to realize the different problem which arises in peripheral nerve injury. After injuries to the peripheral nerves there is usually a serious loss of those afferent stimuli which are of such importance for the perfect performance and adjustment of the finer and more delicate movements, and consequently in the later part of the treatment every effort must be made to develop those purposive and more complex movements which the particular patient will require when he returns to a civil occupation.

(*b*) *Explanation of charts.*—The vertical columns, 1 to 24, refer to the months after the performance of the suture; in the last column, "delay," reference is made to any case where an interval of eighteen months or more has occurred between the date of the reception of the wound and the time of the operation, the figures express this interval in months. In all the charts reference is made, unless otherwise stated, to the return of voluntary power, since in my experience this has been more frequently discovered at an earlier date than the recovery of faradic response. The muscles are recorded by means of the initial letters, viz.:—

SL = supinator longus.

As regards sensation information is conveyed by the following abbreviations and letters:—

P = commencement of recovery of protopathic sensibility.
P.rec. = complete recovery of protopathic sensibility.
E. = commencement of recovery of epicritic sensibility.

The interrupted lines in certain columns mark the date of the last examination, unless they occur in the last column, in which case several of the patients have been under observation for further periods, but the intervals between the later examinations have been longer than four to six weeks.

(c) *Musculo-spiral nerve.*—This nerve offers the best opportunity of investigating many of the problems, since the influence of two factors, "bad shunting" and the loss of afferent impulses from joints, &c., is reduced to a minimum, and of much less significance than in such nerves as the median or ulnar, which contain a large proportion of afferent and sympathetic fibres, as well as efferent. This, together with the fact that suture of the musculo-spiral must occur in the proximal part of the limb, may account for the constancy of a good recovery.

The patients may be divided easily into three groups according to the anatomical level of the suture.

(1) In the lower third of the arm, i.e., below the level of the musculo-spiral groove, and where the trunk is lying deeply between the brachialis anticus and supinator longus. In two patients, destruction of the branch to the supinator longus was recorded at operation, and this is responsible for the absence of recovery of this muscle in patients 10 and 18. Excluding the three failures, the recovery has followed a fairly regular course, and the order of the muscles as they regain voluntary power has followed very closely the anatomical arrangement of the branches (fig. 1). In patients 12 and 19 the recovery is divided into two stages by an interval of eight months and ten months respectively, a phenomenon

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 1				S.L.	E C R.		E C.U		E C D.				E O M P	E L P		E B P								-----	
,, 2				S L.		E C R	E.C U	E C D.	E O M P			E.L P												-----	
,, 3					S.L.		E.C R.			E C U				E L P.		E.O.M P	E C D								
,, 4						S L	E C R	E C U.				E C D		-----											
,, 5				S L	E C R	E C U.	E C D	-----																	
,, 6				S L	E.C.R.	3 E C D.	E C U.				1 2 3 4 E.C D	E O M P	E.L P	E B P		-----									
,, 7				S L.	E.C R		E C.U	E.C D			E.O M P		E L P												
,, 8						S.L		E C R					E C U.		E C D.	E O M P			E L P.				E B P	-----	
,, 9			S L		E.C R			E C D			E O M P	E.L.P													
,, 10				E C R.	E.C U	E L.P	E O M P	1 2 E.C D																	
,, 11				S L.	E.C.R		E C.U.	E O M.P						E C D.					E L P.		E.B.P			-----	
,, 12				S L	E C.R		E.C U.									E.C D		E.O.M P		E B P				-- --	
,, 13			S L	E C R.				E C D.																	
,, 14		S L	E C R																						
,, 15				S L	E C R			-----																	
,, 16			S.L.			E.C.R	E.C.U.	E.C.D.			-----														
,, 17											E C R.					E O M P	E L P			-----					
,, 18			E.C.R.		E C U.																				
,, 19								E.C R.	E C U											E.C.D		E O M P.		E L P	
,, 20			E.C.R																						
,, 21			E O M P																						
,, 22				S.L																					
,, 23													Failure							-----					
,, 24												Failure							-----						21
,, 25														Failure								-----			19

CHART I.—Musculo-spiral nerve (lower third of arm).

The numerals above E.C.D. refer to the first (index), second, third and fourth (little) fingers.

which will be found in several of the other charts. A possible explanation is that the young fibrils, which subsequently supply the muscles late to recover, have encountered some obstruction, and the delay is due to the time spent in penetrating this.

Of the three failures, one had previously had a nerve graft inserted to bridge a large defect, which procedure had proved unsuccessful; in the second, prolonged suppuration had prevented surgical interference until twenty-one months after the injury; whilst in the third there is clinical evidence of a neuroma at the site of the suture, indicating that some regeneration has occurred, but for some reason, as yet undetermined in this case, the young fibrils have been prevented from penetrating the distal end.

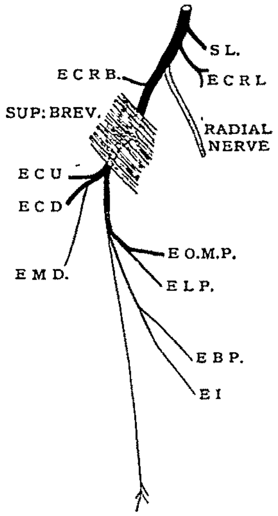


Fig. 1.

(2) The sutures performed in the middle third of the arm, in the musculo-spiral groove, include the four complicated by an ununited fracture of the humerus to which reference has previously been made.

Patient 46 was seen for the first time eighteen months after operation, and there was then no recovery of voluntary power, but a faradic response was present in the supinator longus and the extensors of the wrist and fingers. With suitable treatment, he is recovering from this functional disability.

With the exception of those patients in which an ununited fracture of the humerus occurred, there was only one failure in this series,

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 26				S.L	E.C.R. E.C.U.				E.C.D	E.O.M.P	E.L.P			E.B.P			----								
,, 27				S.L.	E.C.R.	E.C.U.					E.C.D		E.O.M.P												
,, 28					S.L.		E.C.R. E.C.U.			E.O.M.P.		E.C.D				E.L.P.								E.B.P.	
,, 29					S.L	E.C.R		E.C.U.	E.O.M.P																
,, 30						S.L.		E.C.R.			E.C.U.			----											
,, 31									E.C.R		S.L				E.O.M.P						----				
,, 32					S.L	E.C.R		E.C.D	E.C.U		E.O.M.P	E.L.P		E.B.P			----								
,, 33					E.C.R.	S.L.	E.C.U.				----														
,, 34					S.L.	E.C.R.		E.C.U																	
,, 35				S.L.	E.C.R.		E.C.U.		----																
,, 36			S.L	E.C.R		----																			
,, 37					S.L	E.C.R.																			
,, 38				S.L.		E.C.R.																			
,, 39			S.L.			E.C.R.																			
,, 40													E.C.R.		E.C.U.	3 4 E.C.D							----		
,, 41								S.L.	E.C.R.		E.C.U.				E.O.M.P.		E.C.D.		E.L.P.					----	
,, 42				S.L.																					
,, 43																					----	All acting			
,, 44					E.C.R.		----																		
,, 45																				S.L	2½ years E.C.R.	E.C.U.	E.C.D	E.O.M.P	
,, 46												Faradism S.L.	E.C.R.	E.C.U.	E.C.D.			Nil Vol:			----				
,, 47						S.L	E.C.R.					Bone graft went septic —						Failure					----		
,, 48												E.C.R.	S.L						E.C.U.		Ununited Fracture				
,, 49																	Failure				Ununited Fracture				
,, 50														----		Failure		—	Ununited			Fracture			
,, 51																		Failure			(2 yrs.)		Bulb.	----	24

CHART II.—Musculo-spiral nerve (middle third of arm).

and in this patient an interval of two years had elapsed before the suture was performed.

(3) The rapidity and completeness of recovery in the first four patients on Chart III is of importance and interest, since the suture in each was above the level of the groove, and may be considered as being in the axilla.

Patient 56 collapsed during the operation, and the ends of the nerve consequently were merely anchored by one through-and-through stitch, yet voluntary power has slowly returned in the supinator longus and extensors of the wrist.

Chart IV demonstrates the date of recovery of voluntary power in the various muscles at the three levels, and reference to it shows that recovery is proportionately more rapid the nearer the suture is to the spinal cord. In the lower third of the arm the supinator longus recovered most frequently about the fourth month, whilst in the upper third about the seventh month, yet the regenerating fibrils in the latter had to grow more than three times the distance traversed in the former. Similar results are to be seen in the other muscles. Are we to conclude from this that a nerve fibre regenerates more rapidly and readily the nearer the section has been made to its anterior cornual cell? The better prognosis for sutures in the upper arm compared with those in the forearm supports such a contention.

From the foregoing results it may be concluded that a good functional result is to be anticipated after secondary suture of the musculo-spiral nerve, provided there is no special complication or contra-indication.

The extensors of the wrist usually re-

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Patient 52					S.L.	E.C.R.	E.C.U.	E.C.D.			E.O.M.P.	E.L.P.			E.B.P.									
" 53							S.L.	E.C.R.	E.C.U.									E.O.M.P.	E.L.P.	E.C.D.				
" 54							S.L.		E.C.R.		E.C.U.	E.O.M.P.					E.C.D.			-----				
" 55							S.L. E.C.R.		E.C.U.	E.O.M.P. E.B.P.		E.C.D.		E.L.P.							-----			
" 56			Triceps							S.L.			E.C.R.			E.C.U.	-----							
" 57						S.L.	E.C.R.	-----																
" 58						E.C.R.	-----																	

CHART III.—Musculo-spiral nerve (upper third of arm).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.		1.	3.	9.	1.	2.																		
E.C.R.			3.	2.	8.	2.	2.	2.			1.													
E.C.U.					2.	1.	7.	1.	1.	1.			1.											
E.C.D.						1.	1.	6.	1.			1.		1.	1.	1.	1.			1.				
E.O.M.P.			1.				1.	1.	1.		2.	1.	1.			3.		1.				1.		
E.L.P.						1.						2.	2.	2.			1.		2.	1.				1.
E.B.P.														1.		1.					1.		1.	

Lower third (twenty-five cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.			2.	5.	5.	3.		1.			1.		1.											
E.C.R.				1.	5.	6.	2.	1.	2.			1.	1.											
E.C.U.					1.	1.	3.	2.	1.		2.				1.				1.					
E.C.D.								1.	1.		1.	1.				1.	1.							
E.O.M.P.									1.	2.	1.		1.		2.									
E.L.P.											1.	1.				1.			1.					
E.B.P.														2.										1.

Middle third (twenty-six cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.					1.	1.	3.			1.														
E.C.R.						2.	2.	1.	1.				1.											
E.C.U.							1.		2.		1.					1.								
E.C.D.								1.				1.				1.				1.				
E.O.M.P.										1.	1.	1.						1.						
E.L.P.												1.		1.					1.					
E.B.P.											1.				1.									

Upper third (seven cases).

CHART IV.—To show date of recovery of voluntary power of the various muscles at the three levels.

gain full power, and the extensor communis digitorum recovers satisfactorily, although the extension of the index is not infrequently imperfect, and it is almost the rule to find that the patient is unable to extend this finger alone at the metacarpo-phalangeal joint.

The extensors of the thumb rarely show such a perfect recovery, the extensors brevis pollicis being most commonly paretic, which is the common cause for the imperfect extension of the thumb and the difficulty experienced in performing such a movement as opening a pair of scissors.

The extensors of the wrist have never been found to recover their synergic action before that of a prime mover; in fact, in end results it has been almost constantly found that the grasp was weak, owing to the loss of the synergic extension of the wrist, unless this movement had been carefully developed during the period of muscle-training. A number of patients have returned to hospital complaining of weakness of the hand grasp, although the nerve had regenerated well and the extensors of the wrist were quite powerful, and this has been found to be due simply to the loss of their synergic action. It is hardly to be expected that there would be a spontaneous recovery of the synergic action unless the same set of fibres innervated the extensors of the wrist after the suture as before, a condition which is not likely to occur frequently.

(*d*) *Median nerve.*—Unfortunately many patients, in whom this nerve had been sutured at the wrist, have been lost, as they were able to procure light employment, and it was not often possible to keep them under observation for long periods. In quite a large proportion only an incomplete recovery of protopathic sensibility has occurred, and voluntary power in the abductor brevis pollicis has only been found in some 40 per cent. of those in which sufficient time has elapsed to expect this to be possible.

Only the abductor brevis pollicis of the thenar group has been mentioned, because the flexor brevis pollicis is composed of several slips, some supplied by the median and others by the ulnar, which are subject to considerable individual variation; and it is consequently difficult to give a reliable opinion as to the presence of voluntary power in the flexor brevis pollicis from a clinical examination.

Of the sutures in the forearm (Chart VI) in patients 82 to 90, this was performed below the level of the anterior interosseous branch; in two (patients 91 and 92) above this branch, but distal to the supply to the flexor carpi radialis and pronator radii teres; whilst in the

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 59				P.							Prec							A.B P.						-----	
,, 60					P.									Prec.	A.B.P.	E.		-----							
,, 61					P.							Prec.			E.			A.B P.						-----	
,, 62					P.			Prec.		-----															
,, 63					P.		A.B.P.				-----														
,, 64								P.	A B.P.		Prec.					E.				-----					
,, 65			P.					-----																	
,, 66				P.					-----																
,, 67				P.				Prec.			-----														
,, 68				A.B P.	P.							-----													
,, 69				P.											-----										
,, 70				P.		A B.P.				Prec.		-----													
,, 71			P.	A B.P.						-----															
,, 72				P.						-----															30
,, 73				P.								-----													
,, 74			P.			-----																			
,, 75			P.			-----																			
,, 76			P.			-----																			
,, 77					P.												-----								
,, 78				P.								Prec.													
,, 79					P.								-----												19
,, 80																		Failure							
,, 81																		Failure							18

CHART V.—Median nerve (wrist).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 82			$\overset{1}{\text{F.S.D.}}$	P.			-----																		
,, 83				$\overset{1}{\text{F.S.D.}}$	P.							A.B.P.													
,, 84				A.B.P.	$\overset{1}{\text{F.S.D.}}$				P.				E.				-----								
,, 85					$\overset{1}{\text{F.S.D.}}$						P.	A.B.P.												-----	
,, 86				P.		$\overset{1}{\text{F.S.D.}}$		-----																	
,, 87			P.	$\overset{1}{\text{F.S.D.}}$					-----																
,, 88						P.		$\overset{1}{\text{F.S.D.}}$																	20
,, 89							$\overset{1}{\text{F.S.D.}}$		P.					-----											
,, 90		P.			$\overset{1}{\text{F.S.D.}}$																				
,, 91				F.S.D.	P.			F.P.D.		F.L.P.		E.		-----											
,, 92							P.	F.L.P.		F.S.D.	F.P.D.	Prec.			A.P.B.			E.						-----	
,, 93				F.S.D.	P.R.T.			F.L.P.	P.						Prec.										
,, 94				F.C.R.	$\overset{2\,3.4.}{\text{F.S.D}}$	P.			F.L.P	F.P.D								$\overset{1}{\text{F.S.D.}}$						-----	
,, 95				F.L.P.														F.S.D.						-----	
,, 96			P.	F.S.D.														-----							41
,, 97										P.			F.C.R. F.L.P				P.R.T.			-----					
,, 98			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 99					P.R.T.	F.C.R.					P.	-----													
,, 100						F.S.D.					F.L.P.		-----												20
,, 101										P.		-----													
,, 102				P.		-----																			42
,, 103																	All acting vol: –				3½ years Anaesth: – only index				
,, 104																	All acting (except F.S.D.) –				2½ years Anaesth: distally				
,, 105																	All muscles acting –				2½ years Anaesth: 2 fingers				
,, 106															Nil vol:*		F.C.R. – F.S.D. – F.P.D. –				resp: to Farad:				
,, 107																		Failure							

Chart VI.—Median nerve (forearm).

Patients 82 to 90 below anterior interosseous branch; patients 91 and 92 above anterior interosseous, but below branches to flexor carpi radialis and pronator radii teres; patients 93 to 107 above all muscular branches. Numerals above flexors of fingers refer to first (index), second, third and fourth (little) fingers.

* Date when first examined after suture.

remainder it was done proximal to all the muscular branches. In several of the latter group, branches, in addition to the nerve trunk, were injured, and this was responsible for the failure of recovery of some of the muscles.

Patients 103, 104, and 105 are later results, and were first examined some considerable time after the suture.

Patient 106 is another example of a hysterical loss of power; three of the muscles responded to faradism, and there was a sensory loss of the "glove" type.

The results in Chart VII are comparable to the satisfactory recoveries already seen after suture of the musculo-spiral in the proximal part of the limb as regards completeness, regularity, and rapidity of recovery.

The late commencement of improvement and the slower progress in patient 116 is probably due to the ligature of the axillary artery, since no other reason for this can be determined.

(*e*) *Ulnar nerve.*—The disappointing results which follow suture of this nerve are now well known, but it is still difficult fully to understand the reason for the poor prognosis. The risk of "bad-shunting" is clearly considerable, and may be the chief explanation; and from this it is easy to understand how the intrinsic muscles of the hand, with their small nerves of supply, run a greater risk of not receiving any efferent fibres than the larger muscles. At one time it was imagined that a poor blood supply might be the cause of the incomplete recovery or failure; but, from dissections made after special injection of the blood-vessels of the limb, I find that the ulnar nerve has quite as efficient, and often a richer, blood supply than the median.

Many of the patients, in which the suture was performed in the forearm, have been lost owing to the reason mentioned in the case of suture of the median at the wrist.

In three out of the seven failures in Chart VIII it is of interest to notice that there was a delay of more than eighteen months before the operation was done.

After suture in both the arm and the forearm the recovery of voluntary power in the small intrinsic muscles of the hand was particularly poor and disappointing.

Brouwer [1], in a recent paper, advances a hypothesis which may prove to be a true explanation of the bad prognosis for the intrinsic muscles after lesions of the median and ulnar nerves. He found, in neuritis of the median, that in a large number of patients there was a

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 108						P.R.T.	F.C.R.				P.	F.L.P.		F.S.D.				F.P.D.			A.B.P.	Prec.		----	
,, 109					P.R.T. F.C.R.	P.		^{2,3,4}F.S.D.						^{1}F.S.D.	Prec.					----					
,, 110					P.R.T.	F.S.D. P.	F.C.R.							F.L.P.	Prec.	A.B.P.					F.P.D.			----	
,, 111				P.	P.L.	F.C.R.	F.S.D.	F.P.D.		----															
,, 112				P.		^{2,3,4}F.S.D.	^{1}F.S.D.								F.C.R.		----								
,, 113					P.	P.R.T.	F.C.R.	^{2,3,4}F.S.D.					----												
,, 114					P.R.T. F.C.R.	F.S.D.	F.L.P. P.	F.P.D.																	
,, 115						P.R.T. F.C.R.					P.	F.L.P. F.S.D.						F.P.D.						----	
,, 116							P.R.T.				F.S.D.	F.L.P.	F.C.R.												
,, 117					P.R.T.	F.S.D.		F.L.P.										F.P.D.		A.B.P.				----	
,, 118				P.		P.R.T.	F.C.R.																		
,, 119			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 120			P.R.T.		P.		F.C.R.	F.S.D.	A.B.P.								Prec.								
,, 121							P.R.T.	F.C.R.	^{2,3,4}F.S.D.			----													
,, 122				P.		P.R.T.	F.C.R.	F.S.D.		Prec.															
,, 123			P.R.T.	P.		^{2,3,4}F.S.D.		^{1}F.S.D.			F.L.P.														31
,, 124					P.																				
,, 125																			P.R.T.	- P.L.	F.C.R.	- F.S.D.	- *F.L.P.	- Prec.	
,, 126																P.R.T.	- F.C.R.	- F.S.D.		- *F.P.D.	- Prec.	- E.		----	19
,, 127														P.R.T.	- P.L.	- F.C.R.	- F.S.D.		- *F.L.P.	- A.B.P.	- Prec.				
,, 128												P.R.T.	- F.C.R.		*F.S.D.	- F.L.P.									
,, 129			P.R.T.	F.C.R.						F.S.D.	F.L.P.		----												
,, 130							F.C.R.	----																	

CHART VII.—Median nerve (arm).

* Date when first examined after suture.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 131							P.							A.M.D.		----									
" 132						P.					P.rec.		A.M.D.									E.		----	
" 133						P.				A.M.D.		E.			----										
" 134			P.			A.M.D.		P.rec.	Inter																
" 135							P.						----												
" 136							P.	A.M.D.																	
" 137			P.			A.M.D.	P.rec.			Inter.										----					
" 138															A.M.D.	P.	Inter.								
" 139								P.																	
" 140						P.												P.rec.			E.	----			
" 141					P.		----																		
" 142					P.	A.M.D.																			
" 143			P.				----																		
" 144						P.			----																
" 145					P.	----																			
" 146				P.				----																	
" 147				P.				----																	21
" 148					P.																				
" 149								P.						----											
" 150					P.					----															22
" 151				P.		----																			18
" 152						P.						P.rec.			----										
" 153																				A.M.D.	only.		3 years		
" 154																				A.M.D.,-	Inteross:	No Sens: rec.			

9 No evidence of regeneration 6 - 10 mths.

7 Failures

CHART VIII.—Ulnar nerve (forearm).

Distal to branches to flexor carpi ulnaris and flexor profundus digitorum.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 171							P.		A.M.D.											Prec.				-----	
" 172			F.C.U.	F.P.D.	P.		A.M.D.		-----																
" 173			F.C.U.	F.P.D.		P.								-----											
" 174				P.						---															
" 175				F.C.U.			F.P.D.				P.													-----	
" 176						F.C.U.																			
" 177						F.C.U.														P.			A.M.D.		
" 178				F.C.U.									F.P.D.				A.M.D.							-----	
" 179						F.C.U.																			
" 180			F.C.U.						F.P.D.							-----									
" 181			F.C.U.			-----																			
" 182								P.																	
" 183		F.C.U.			F.P.D.			A.M.D.																	
" 184											F.C.U.	P.	F.P.D.									Prec.			
" 185				F.C.U.	P.	F.P.D.																			
" 186			F.C.U.		F.P.D.			A.M.D.		-----															
" 187						P.					-----														18
" 188									F.C.U.																
" 189				F.C.U.	F.P.D.																				
" 190				F.C.U.	P. F.P.D.					A.M.D.		-----													
" 191				F.P.D.								-----													
" 192				F.P.D.	P.																				18
" 193		F.C.U.	F.P.D.		P.								-----					-----							
" 194			F.C.U.	F.P.D.		----																			
" 195						F.C.U.		F.P.D.					----												
" 196			F.C.U.										----												
" 197				F.C.U.			----																		
" 198			F.P.D.	P.																					
" 199				F.C.U.				----																	
" 200																						F.C.U.	only	*	
" 201																			F.C.U.	& *	F.P.D.	only			
" 202																	F.C.U.	& * F.P.D.	only						
" 203																		F. C. U.	* only						
" 204																		F.C.U.-F.*	P.D. &	Inteross:	Prec.				18
" 205																				F.C.U. -	F.P.D. -	A M D.	&	P.*	
" 206																				F.C.U. &	F.P.D.	only	3½	years	
								8	Failures		& 2	nil	at	6 months											

CHART IX.—Ulnar nerve (arm).

loss of voluntary power and faradic response in the thenar muscles—although the other muscles supplied by the median were perfectly normal, and ascribed this greater vulnerability of the muscles of the thumb, or their nerve branches, to the "finer construction of the thumb and higher functional significance which it has received in the phylogenesis." Whether Brouwer's suggestion is the sole or part explanation, or whether the infrequent recovery of the intrinsic muscles is due to their distance from the cells of the anterior cornu, future experimental and clinical research must finally decide, but reference to the charts and consideration of the recovery of afferent, as well as efferent, fibres suggests that there is evidence that the latter is, at any rate, a factor.

The complete separation of the extensor brevis pollicis from the extensor ossis metacarpi pollicis is a human characteristic, and since the extensor brevis pollicis was found to recover less frequently than either of the other extensor muscles of the thumb after suture of the musculo-spiral, it would appear, at first sight, that this was further support of Brouwer's contention that more recent acquisitions are more vulnerable than ancient ones. But this support cannot be accepted without qualification, because the extensor brevis pollicis is innervated at a much lower level than the other two muscles, and its nerve of supply is considerably smaller than either that to the extensor ossis metacarpi pollicis or the extensor longus pollicis (fig. 1); and, furthermore, it is significant that the extensor brevis pollicis recovered in 16 per cent. of all the sutures in the lower third of the arm, 17 per cent. in the middle third, and in 29 per cent. in the upper third, which figures strongly suggest that the distance of the suture from the spinal cord exerts an important influence upon the prognosis.

(*f*) *Sciatic nerve.*—In this group the one instructive point to direct attention to is that in one patient only, as yet, has there been recovery of the tibialis posticus and the flexors of the toes, although the gastrocnemius, which has a much larger nerve supply, almost invariably recovers, even more readily than any of the muscles supplied by the external popliteal segment.

Patient 247 is an example of the two-stage operation, and after an interval of eleven months there is no clinical evidence of any regeneration.

(*g*) *External popliteal nerve.*—Five failures out of twenty-one is rather a surprising proportion for the external popliteal, as the prognosis after suture of this nerve has often been considered almost as favourable

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 217											Gastr:			E.L.D.	Peron:	T.A.		-----							
„ 218								P.	Gastr:	T.A.		-----													
„ 219				P.										Gastr:											
„ 220													Gastr:		T.A.		E.L.D.			Peron:				-----	
„ 221												T.A.								Peron:	Gastr:		-----		
„ 222																P.		Gastr:						-----	
„ 223									Gastr:	T.A.												All	acting	2½ yrs.	
„ 224														Gastr:		-----									
„ 225					Gastr:												P.						27 mths. Same		
„ 226						Gastr:	T.A.					E.L.D.			P.										
„ 227					Gastr:	P.				T.A.							E.L.D.	-----							
„ 228							P.	Gastr:	T.A.	E.L.D.		-----													
„ 229					P.							Gastr:	T.A.	-----											
„ 230										Gastr:											P.			-----	
„ 231					P.			Gastr:	T.A.		-----														
„ 232				P.		T.A. Gastr:	E.L.H.																		
„ 233								Gastr:			-----														
„ 234					Gastr:	P.					Prec.														
„ 235					P.										Gastr:			T.A.			-----				
„ 236						P.		Gastr:																	
„ 237					Gastr:			T.A.	-----																
„ 238								Gastr:					-----												
„ 239													-----												
„ 240				P.		-----																			
„ 241				P.	Gastr:						-----														24
„ 242				P.			-----																		30
„ 243																						Gastr:	2½ years only		
„ 244																				Failure					
„ 245																						Failure			
„ 246																						Failure			
„ 247											-----														

CHART X — Sciatic nerve.

Φ16264

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	Delay
Patient 248						P.		T.A.				E.L.D.	Peron:													Prec.	
,, 249											P.		T.A.		Peron:						----						
,, 250					T.A.										P.	Peron:	E.L.D.	E.L.H.								----	
,, 251							T.A.	E.L.D.																----			
,, 252				P.		T.A.	Peron:					E.L.D.								Prec.						E.L.H.	
,, 253						P.							----														
,, 254				P.		T.A.							Prec.	----													
,, 255						T.A.	Peron:																				
,, 256										P.					Peron:		Prec.								T.A.	----	
,, 257			P.		T.A.	E L D	Peron:		Prec.					E L H				E.						----			
,, 258							----																				
,, 259							----																				
,, 260													----														27
,, 261																					Nil vol:		All respond to Farad:		*		
,, 262										----																	
,, 263						T.A.		P.		Peron:			E.L.D.		----												
,, 264				Peron:										----													
,, 265																				Failure							
,, 266																							Failure		2 years		
,, 267																								Failure	3 years		
,, 268																					Failure		2 years				
,, 269																							Failure		2 yrs.		

Chart XI.—External popliteal nerve.

as that of the musculo-spiral. An explanation of this, however, is forthcoming when we are reminded of the difficulty of procuring a good bed for the nerve in the region of the head of the fibula, and consequently it is suggested that secondary adhesions and involvement of the nerve in scar tissue are responsible for such a very high proportion of failures.

Patient 261 is a third example of a functional loss of power, although there is good evidence of innervation of all the muscles supplied by the external popliteal.

A comparison of the very rapid and complete recovery of patient 257, with that of the others, is of considerable interest, because his may be considered a primary suture, since only a very slight laceration was found at operation. The recovery, two years after suture, was practically perfect.

(*h*) *Internal popliteal nerve.*—In both patients the functional result has been satisfactory, but in neither has there been any recovery of sensation.

III.—CONCLUSIONS.

(I) The following appear to be the most important factors which may influence the prognosis:—

(1) Sepsis.

(2) Site of the suture, the prognosis being more favourable the nearer the suture is to the spinal cord.

(3) A delay of more than eighteen months before the suture is performed in the distal part of a limb prejudices the chances of recovery, but is not of so much consequence in the proximal part.

(4) "Bad-shunting" is probably a common cause of imperfect recovery in such nerves as the ulnar.

(5) Destruction of branches of the nerve, in addition to injury to the trunk, may be a cause of incomplete recovery.

(6) Ununited fractures, especially of the humerus in injuries to the musculo-spiral, are a serious complication. Evidence appears to be in favour of primary fixation of the bone.

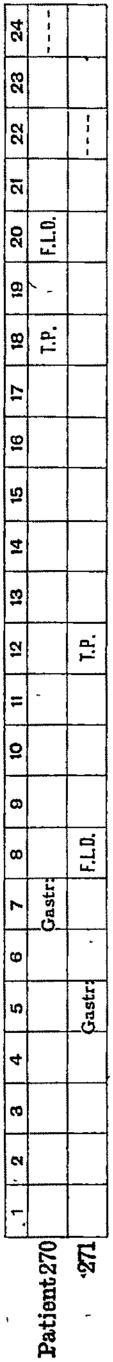


CHART XII.—Internal popliteal nerve.

(7) Ligature of the main artery in the proximal part of a limb may delay and limit recovery.

(8) Imperfect recovery of afferent fibres from such structures as joints, muscles and tendons, is probably an important cause of limited functional recovery of the hand for the performance of purposive actions, although the voluntary power of individual muscles may be good.

(II) The intrinsic muscles of the hand and the muscles in the distal part of a limb recover less frequently than the larger muscles in the proximal part of a limb. Probably several factors contribute to the cause of this :—

(1) Distance which the young nerve fibrils have to travel away from their trophic centre in the spinal cord to reach the muscle.

(2) The size of the nerve of supply. The larger the nerve the greater the chance the young fibrils must have of reaching the muscle.

(3) Possibly the phylogenetic history of the muscles, since there does seem to be some support of the hypothesis that more recent acquisitions are the more vulnerable.

(III) The following points bearing on the surgical technique can be deduced :—

(1) It is advisable to avoid the through and through suture, whenever practicable, as proved also by the experimental work of Sargent and Greenfield [2].

(2) The construction of a new bed for the nerve, after suture, is preferable to any autogenous or heterogenous protective material. The unsatisfactory bed after suture of the external popliteal nerve, in the vicinity of the head of the fibula, is probably a cause of many failures.

(3) Displacement of the ulnar nerve in front of the internal condyle of the humerus or widespread freeing to procure apposition of the ends does not appear to retard the recovery.

(4) Liberal resection of the injured ends is essential and particularly necessary if sepsis is prominent.

(IV) No recovery after the performance of secondary suture could be regarded as perfect. The chief causes of the imperfect result after *secondary* suture appear to be :—

(1) The fact that an appreciable portion of the nerve trunk has to be resected causes inevitably a disturbance of the intraneural anatomy, however carefully the surgical procedure is carried out.

(2) After secondary suture the nerve is making a *second* effort to regenerate, the first attempt being represented by a neuroma or outgrowths along fascial planes.

(3) The almost constant occurrence of one or more of the foregoing factors which have been found to influence the prognosis.

REFERENCES.

[1] BROUWER, B. "The Significance of Phylogenetic and Ontogenetic Studies for the Neuropathologist." *Journ. of Nerve and Mental Dis.*, February, 1920, vol. li, p. 113.

[2] SARGENT, P., and GREENFIELD, J. G. "An Experimental Investigation of Certain Materials used for Nerve Suture," *Brit. Med. Journ.*, September, 1919, vol. ii, p. 407.

MICROGYRIA AND ITS EFFECTS ON OTHER PARTS OF THE CENTRAL NERVOUS SYSTEM.

BY THE LATE W. PAGE MAY, M.D., D.Sc., F.R.C.P.
Fellow and Lecturer of University College, London.
(*From the Pathological Laboratory, London County Asylums, and from the Physiological Laboratory, University College, London.*)

MICROGYRIA, which has hitherto been usually known under the name of hemiatrophy of the brain or arrested development of the nervous system, has been comparatively rarely described, and merits further study.

The following case came ultimately into the hands of Dr. Mott, to whom I would here cordially express my thanks for kindly allowing me to investigate the material and for many valuable suggestions during the course of the work.

SUMMARY OF THE CASE.

Vascular lesion of the right cerebral hemisphere involving chiefly the right centro-parietal region of the cortex with portions of the frontal and temporal lobes and portions of the right basal ganglia. Atrophy and arrested development of the right pyramidal tract, right mesial filet and associated structures in the mid-brain, pons, medulla and spinal cord. Atrophy of the opposite (left) side of the cerebellum with some of its various nuclei and peduncles. Atrophy of the left side of the spinal white and grey matter. Diminution in number of motor cells chiefly in the cervical and lumbar enlargements.

CLINICAL HISTORY.

Since earliest life the patient had suffered from left hemiplegia and mental hebetude. The mental deficiency steadily increased and became early associated with unilateral and epileptiform convulsions of the ordinary type. Sight and hearing were normal but the above symptoms progressed and terminated fatally in his thirty-sixth year.

Brain.—In the brain the coarse deviations from normal are best seen by a reference to the accompanying photographs. There is a remarkable difference in the size of the two hemispheres, the right being about two-thirds as large

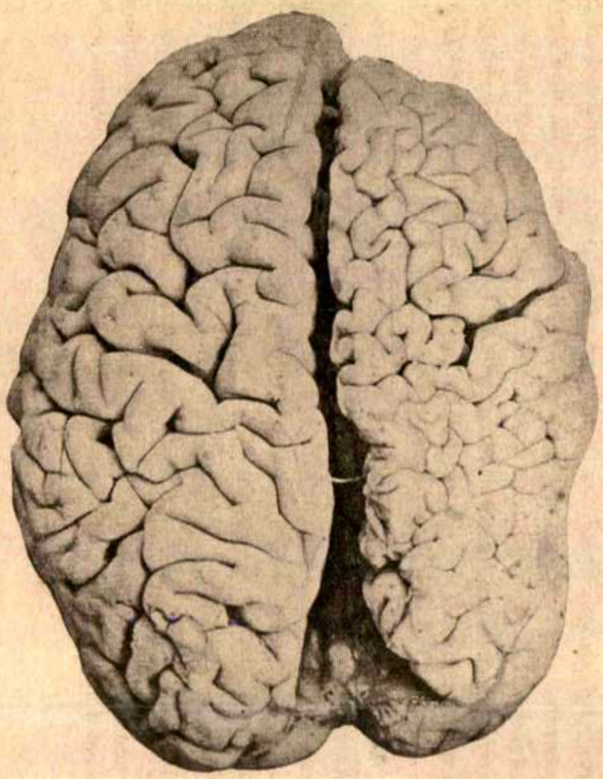


FIG. 1.

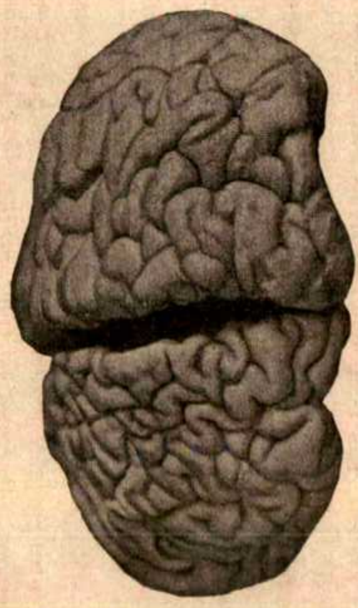


FIG. 2.

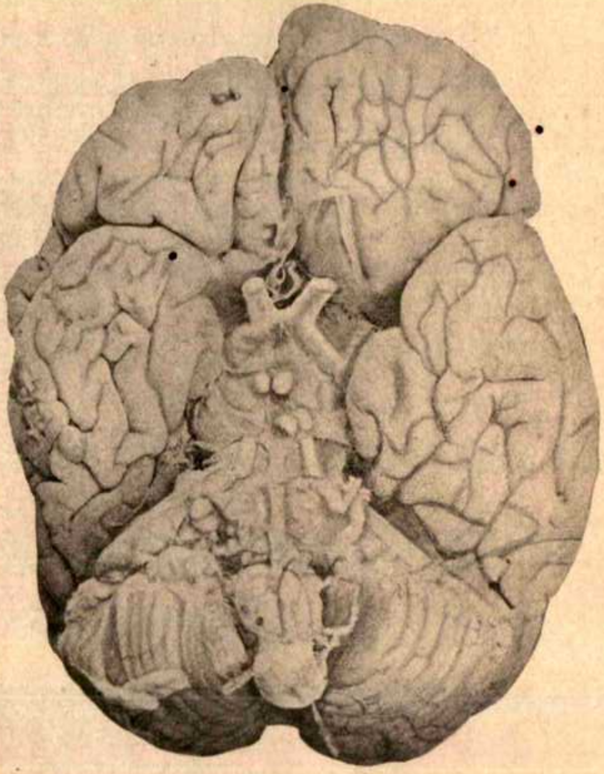


FIG. 3.

as the left, which is normal. The stress of the disease has fallen chiefly on the centro-parietal region of the right side, less on the frontal and temporal and least on the occipital lobe. The convolutions in the affected areas are shrunken in all three dimensions, i.e., they are shorter, narrower and less deep than normal and wanting both in grey and white matter. The corpus callosum is thinner than in health, but the olfactory lobes, optic tracts and cranial nerves are unaffected.

The mode of investigation adopted was to remove pieces of certain selected areas of the cerebral cortex, cerebellum and spinal cord and to stain them by various methods for minute microscopic examination of the cells and fibres. The remainder of the brain and portions of the spinal cord were cut in serial section and stained by the Weigert-Pal process, certain of these sections being again counterstained by alum, cochineal or other suitable colouring matter.

Microscopic examination of the cerebral hemisphere showed that everywhere in the affected region the cortex is thinner than normal and this most of all in the right ascending frontal convolution. All the cortical layers are wasted, but especially the pyramidal and fourth layer.[1] In the leg and body areas the very large, so-called Betz cells are not only much diminished in number and in size but have fewer processes than in health and all of them present some degenerative change. In several sections of this region, only one group of moderately good Betz cells could be found. These were situated on the flat external surface of the right ascending frontal convolution (not in the depth of the Rolandic fissure), and even they were stunted and had fewer processes than natural and showed other signs of degeneration. The right arm area presented much the same appearance as above but the pyramidal layer was not quite so much affected. In the corresponding regions of the left cerebral cortex the histological appearances were normal. The pyramidal and Betz cells were abundant and of healthy structure and no signs of hypertrophy or compensatory overgrowth was traceable. In the right facial area all layers are thin and there are very few large cells in layer 4, whereas the left facial area seems normal. In the right ascending parietal convolution all layers are much wasted except the granular layer, which looks healthy. The right first frontal gyrus has less than half its normal depth and the pyramidal layer is especially poor, whereas the left first frontal is normal. The temporal convolutions are histologically fairly normal, but the pyramidal layer is distinctly better seen in those of the left side than of the right. The visuo-sensory area in the calcarine region of the occipital lobe presents no definite abnormal change on either side but in the right visuo-psychic area the pyramidal layer is distinctly poorly developed.

---

[1] The classification of the layers of the cerebral cortex here adopted is that given by Mott [1], at a meeting of the Ophthalmological Society of London, Nov., 1904.

With regard to the changes in the nerve fibres of the cerebrum, it is noteworthy that the incidence of the disease is chiefly on the efferent projection fibres of the affected side, which are much diminished in number, and present signs of wasting and sclerosis. The tangential fibres of the cortex and the fine network in the inner line of Baillarger

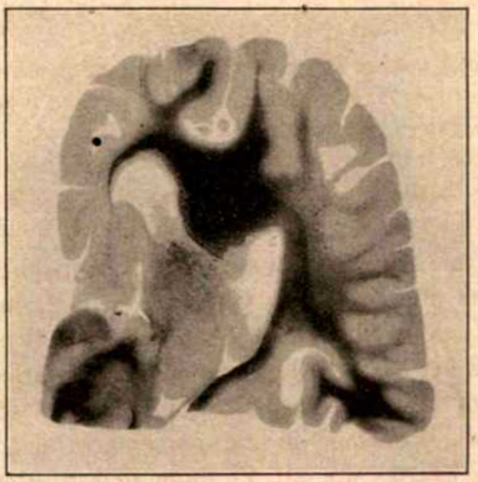


FIG. 4.

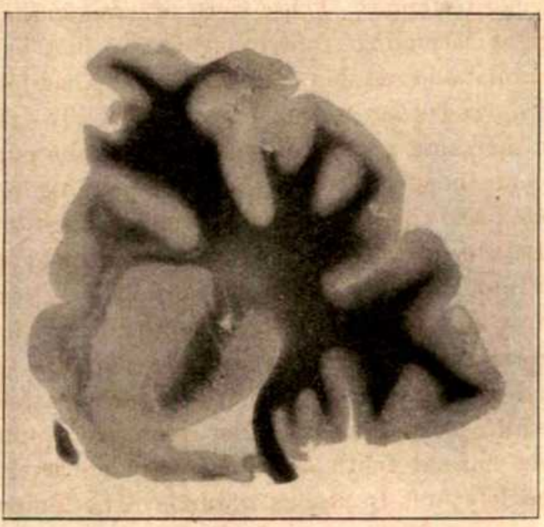


FIG. 5.

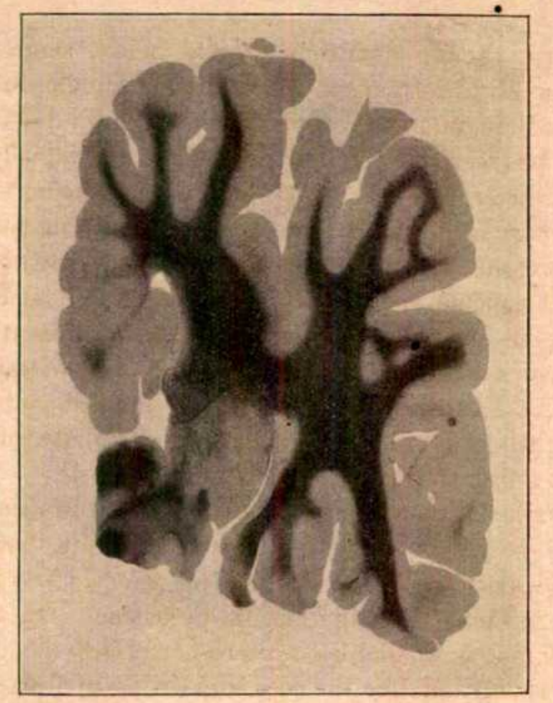


FIG. 6.

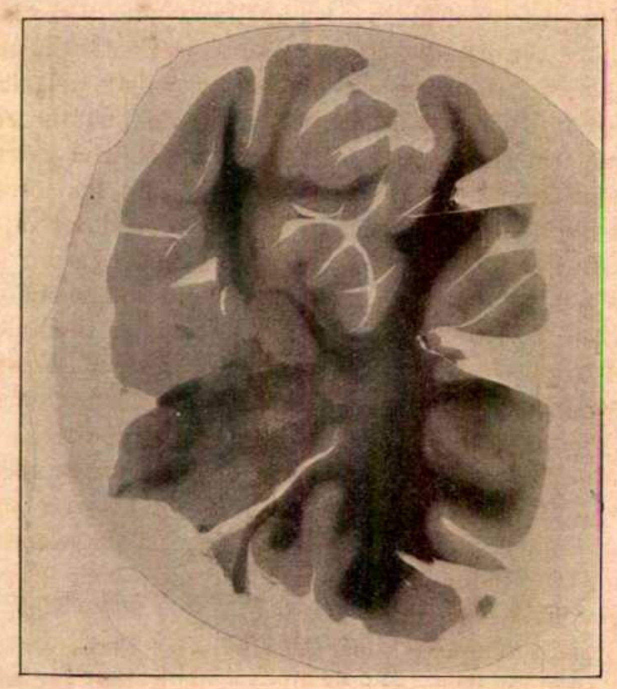


FIG. 7.

are also slightly though definitely affected in the same way. On the other hand, the fibres forming the associational bundles are not at all, or, if at all, scarcely to an appreciable extent.

In the opposite (left) cerebral hemisphere, as already remarked, no compensatory increase of cells could be found, and the nerve fibres seemed in every way normal in number and appearance. The corona radiata on the right side is much smaller than on the left (roughly about half). The right internal capsule is not only much reduced in size in its fronto-caudal plane, but also laterally where its posterior limb (the limb chiefly affected) measures from 2½ to 3 mm., as compared with 5 to 5½ mm. for the healthy side. In its vertical dimensions the right internal capsule is only very slightly reduced. It contains few medullated nerve fibres, and shows all the ordinary signs of atrophy and sclerosis.

The corpus striatum presents only very slight deviation from normal. The nucleus caudatus seems healthy throughout, but the lenticular nucleus, and especially the globus pallidus, are slightly wasted and not quite so rich in fibres as on the normal (left) side. On the affected side the optic thalamus is smaller both antero-posteriorly (i.e., fronto-caudally) and laterally than should be the case, and owing to a marked diminution in the number of its nerve fibres its nuclei are all less distinct than normal. The paucity of fibres is especially striking in the lateral nucleus of the right as compared with that of the left optic thalamus. Moreover, though the right arcuate nucleus is fairly well delineated the "centre-median" of Luys is scarcely traceable, and the red nucleus is less than half the size of that of the right side, and extremely poorly provided with fibres. On the left side the optic thalamus is healthy, and all its nuclei are well seen, notably the dorsal, lateral and median nuclei, the centre of Luys, nucleus arcuatus, and red nucleus and corpus subthalamicum, all of which are well seen and differentiated.

The island of Reil on the right side is smaller than normal, and its convolutions imperfectly developed. The claustrum, together with its capsula externa on its inner side, and its capsula extrema on its outer side, are well marked but slightly thinner than on the normal side. Moreover, the two capsules diminish in thickness from above down (i.e., dorso-ventrally), so that in their lower halves they are scarcely represented, and their junction below at the base of the island of Reil with the inferior longitudinal bundle and the medullary fibres around the anterior commissure is not traceable.

In the right temporal lobe the medullary fibres are generally less numerous than on the normal side, though the auditory radiations extending as a large bundle of well stained fibres from the lateral margin of the middle geniculate body to the first temporal convolution are well marked. Similarly the general wealth of fibres in the occipital lobe is less than on the normal side, yet the optic radiations extending from the lateral geniculate body around the posterior descending horn of the lateral ventricle to the visuo-sensory area of the calcarine region are also well marked. Moreover, standing out in marked contrast to the remainder of the fibres of the temporo-occipital lobe, is the inferior longitudinal bundle which has abundant well stained fibres forming in these transverse (frontal) sections a well defined sickle-shaped mass which has its broader end above at the base of the island of Reil, and curves externally around the descending horn of the lateral ventricle, tapering away to a fine point of fibres in the region of the nucleus amygdala.

It will be seen on reference to the accompanying photographs that the third ventricle and the lateral ventricles, with their cella media, are moderately distended. This porencephalic condition still further reduces the space, which in the left hemisphere is occupied by healthy brain tissue.

Midbrain.—On the right side there is marked atrophy of the pyramidal, and less so of the middle fillet fibres. The red nucleus, as previously mentioned, is smaller than normal, and the perpendicular fibres of the crus, especially those lateral to the corpus subthalamicum, are distinctly less numerous than in the healthy hemisphere. The lateral fillet, the middle and lateral geniculate body, the anterior and posterior corpora quadrigemina on the affected side present no deviations from normal.

Pons.—The tegmentum on the right side is smaller than on the left and the middle fillet fibres are here contrasting with the middle fillet in the medulla much wasted and form a band about half as deep and not quite so broad as that of the opposite side. Also the longitudinal fibres in the mesial portion of the middle fillet are notably very much diminished in numbers, so that the nucleus reticularis tegmenti seems not only to occupy the position which should be chiefly filled by them, but actually to extend laterally, forming a thin band of grey matter with middle fillet fibres dorsally and ventrally to it. The central tegmental tract and the posterior longitudinal bundle seem slightly smaller on this side than on the left, whereas the fibræ paramedianæ are slightly less marked and the superior cerebellar peduncle distinctly less marked on the left side than on the right. The lateral fillets and lateral pontine bundles are equal on the two sides, and with the above exceptions all the remaining structures and longitudinal systems seem normal in the tegmentum of both sides.

In striking contrast with the wasted (or lesser developed) tegmentum of the affected side is the larger development of the crural portion on that side. This is obviously due to the fact that in contra-distinction to the cerebral hemispheres the right half of the cerebellum is healthy whilst its left half is wasted. For, as is well known on physiological and anatomical grounds, one half of the cerebrum is in closest relationship functionally with the opposite half of the cerebellum. Hence in this case the large mass of transverse pontine fibres which chiefly form the middle cerebellar peduncle on the right side, and is in connexion with the larger right half of the cerebellum, is distinctly bigger than its fellow and displaces the anterior median groove of the pons to the left. It is interesting to note too that in relationship with the changes in the frontal and temporal lobes the temporo-pontine bundles situated in the dorso-lateral angle of the pons are somewhat wasted, and the fronto-pontine fibres situated mesio-ventrally are also wasted, but less so, in keeping therefore with the lesser affection of the main mass of the associated area of the frontal lobe. The right pyramidal bundles are much wasted and to a considerable extent broken up, more so than on the left side, by the transverse fibres of the pons.

Medulla.—Corresponding abnormalities obtain in the right half of the medulla where the pyramidal fibres are greatly diminished in number and some in size. The middle fillet is also smaller than its fellow, though the difference in bulk is not nearly so great as between the middle fillets higher up in the brain stem and notably in the mid-brain. Also, there is a distinct relative smallness on this side (right) of the nucleus lateralis, and of the inferior olive. The internal arcuate fibres are less numerous and also the fibræ comitantes quinti, though the spinal roots of the fifth nerve seem equally well developed. It is to be expected that the inferior olive, which is known to give origin to the system of afferent fibres (olivo-cerebellar) which cross the middle line and terminate chiefly in the opposite half of the cerebellum, should in this case be smaller on the right side because it is thus in connexion with the smaller half of the cerebellum (the left), but it is curious that the internal arcuate fibres should be more numerous on the left side of the medulla than on the right, because after decussating in the middle line they bend forwards (frontally), and to a great extent form the right middle fillet which is smaller than its fellow. It could not be proved that the left posterior column nuclei from which the left internal arcuate fibres almost entirely take origin were larger (or smaller) than the right, yet the fact that the internal arcuate fibres were more numerous on the left side was not only apparent in the medulla, but similarly their homologues throughout the pons were also more numerous and better stained than those on the right. The bundle of Schütz, a network of fine fibres extending from the medulla to the cerebral peduncles and lying just dorsal to the twelfth and similar nuclei, is less evident on the right side than on the left. But, as would be anticipated, the right inferior cerebellar peduncle, formed chiefly by the junction of the right direct cerebellar tract with fibres coming from the larger left inferior olive and passing to the (larger) right half of the cerebellum, is larger on the right side than on the left. The remaining structures in the medulla seemed normal, and call for no comment.

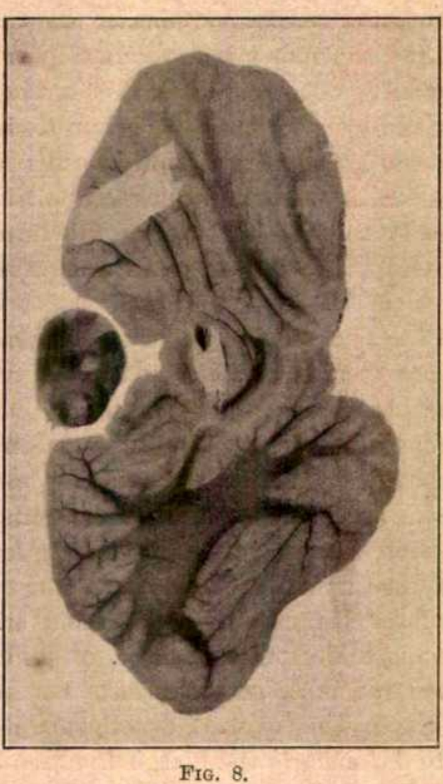


FIG. 8.

Cerebellum.—The gross changes in the cerebellum are best realized by a reference to the accompanying figure. The left half (i.e., the side opposite to the affected cerebrum) is much atrophied. On microscopic examination it is found that in the wasted area the cells of Purkinje are small in number and in size and their processes are much stunted, though here and there a few comparatively healthy cells may be seen. The granular layer, and slightly also the molecular layer, is thinner than in health and seem to be equally affected throughout their depth. An outer layer of round cells, two or three deep, immediately beneath the Purkinje cells, as found by Mott and Tredgold in a similar case, are not in this instance visible. The so-called cells of Golgi seem also distinctly diminished in number in the diseased area. The left nucleus dentatus is smaller and thinner in all dimensions than the right, and evidently associated with this is the fact that the left superior cerebellar peduncle which takes origin from it is smaller than its fellow. The nuclei globosus and emboliformis are slightly smaller and less rich in fibres than on the opposite side, and the left nucleus fastigii (*vel* tecti) is very distinctly smaller, and owing to a paucity of fibres and internuclear fibrillar network is paler and less well marked than on the right side.

The spinal cord.—The spinal cord is profoundly asymmetrical, and though both sides are affected, the left is much smaller than the right—the atrophy of the left side of the cord being in keeping with the arrested development of the right cerebrum and the left side of the cerebellum. The right half of the spinal cord presents no obvious diminution in size except in its anterior column. On microscopic examination it is seen that the right direct pyramidal tract and the left crossed pyramidal tract are much smaller than normal, the number of their fibres is much diminished, and, in addition, many of the fibres present show the ordinary signs of secondary degeneration and sclerosis. Moreover, these processes have not been limited to the above mentioned tracts, but involve also, though to a slighter extent, the deep, and more particularly the marginal, fibres of the left antero-lateral column. Similar slight changes are noticeable in the right crossed pyramidal tract and right antero-lateral column due to affection of some of the homolateral fibres. The posterior columns present no definite abnormalities, but the white fibres of the anterior commissure are in most sections decidedly more abundant on the *left* side than on the *right*. The left anterior column, which, by contrast with the atrophied right, seems almost larger than normal, is very well developed, and has displaced the median longitudinal fissure to the right. The grey matter of the spinal cord is affected on the two sides, and that somewhat irregularly at different levels. The central canal, though not unduly dilated, is here and there multiple, so that two or three small canals lie in contact, each surrounded by the ordinary (healthy) columnar epithelium. The stress of change in the grey matter has naturally fallen on the anterior horns, and on the whole the left anterior horn is smaller, narrower, and more stunted than the right. This is especially seen in the cervical and lumbar enlargements. In a somewhat similar case as this, published by Mott and Tredgold, and previously quoted, the cells chiefly wasted were in the lateral and postero-external groups, but here, though these are to a large extent affected, and in some places practically absent, it is noteworthy that at certain levels the mesial groups, both anterior and posterior, as well as the central group of cells, are almost absent in the left anterior horn. The cells, mostly of smallish size and fusiform, of the lateral process to which Bruce has recently called attention, are well preserved at most levels, and better on the right than left. The columns of Clarke are little affected, but in the lower dorsal region their cells are larger and slightly more numerous on the right side. In all cases, however, these cells show a marginal accumulation of granules, with a peripherally displaced nucleus, that is to say, the ordinary structure of the Clarke-Stilling cells.

REMARKS.

Such cases as described above have usually been known under the name of hemiatrophy of the brain or arrested development of the nervous system. All of them are probably due originally to a vascular lesion, either arterial or venous, occurring in fœtal or early life. The chief organic changes result from arrested development of certain parts of the central nervous system, whilst others continue to grow more or less as in health; hence the title of atrophy or hemiatrophy seems ill-chosen. On the other hand, as a result of arrested development and function in certain structures, others often far distant from the original lesion undergo secondary and tertiary atrophy, and these atrophies may give rise to the most striking organic changes or symptoms in a case, and therefore the term of arrested development of the nervous system or hemiagenesis suggests a distorted idea of the case. On the whole, it seems to the writer that the term microgyria, which involves no causal theory of the disease, and yet at once indicates the grossest change visible to the naked eye in such a case, is better than either of the other two in common usage.

Mott and Tredgold [2] state that these cases readily group themselves into two classes: (1) Cortical, in which the lesion is confined to the cortex or underlying white matter. (2) Basal, in which the original lesion is situated in the structures at the base of the brain; and they describe three cases as illustrating this. Though undoubtedly most of these cases can be thus conveniently grouped, there are occasionally others in which the original lesions may be both in the cortex and in the basal structures of the brain. Judging from the relative amount of disease in the cortical efferent systems and in the structures originating or terminating in the basal ganglia, the writer is of opinion that the case described above falls into an intermediate group of cases, partly cortical, partly basal.

Mott and Tredgold [3] state that "it has been conjectured that a descending thalamo-spinal path exists in man." By reference to two cases, including one of microgyria, and by a lucid deduction from facts. they proved the existence of such a path, and gave photographs of it, It may be of interest in this connexion that the writer, at a meeting of the Physiological Society at Cambridge in August, 1904, demonstrated specimens of the brains and cords of twelve monkeys in which he had caused degeneration in such a descending thalamo-spinal path by production of a localized lesion in the optic thalamus. This atrophy, or degeneration of the thalamo-spinal path, in the case described in this paper, is not (unlike that in the case described by Mott) obtrusive, because, firstly, the relative amount of disease in the thalamus is smaller; and, secondly, the amount of disease in the surrounding structures in the record is relatively greater.

It is not within the scope of this paper to enter into further detail, which, with organic changes in nearly every part of the central nervous system, would take one too far afield. The main fact that the right half of the brain is structurally and functionally chiefly associated with the left half of the cerebellum, and with both sides of the spinal cord, but chiefly the left, finds striking illustration in the present case, and further detail is given in the body of the paper.

REFERENCES.

[1] Mott, F. W. "The Progressive Evolution of the Structure and Functions of the Visual Cortex in Mammalia"—The Bowman Lecture, *Trans. Ophth. Soc.*, January, 1905, vol. 25, p. liii.

[2] Mott, F. W., and Tredgold, A. F. *Brain*, 1900, vol. xxiii, p. 239.

[3] *Ibid.*, p. 248.

A PSYCHOLOGICAL INTERPRETATION OF ESSENTIAL EPILEPSY.

BY L. PIERCE CLARK, M.D.
New York.

I.

As ordinarily considered to-day, epilepsy is recognized by loss of consciousness and a convulsion. Probably epilepsy as a disease has a more ancient lineage than any other nervous disorder. Throughout the ages the nature of epilepsy has been, and still is, the subject of surmise, conjecture and scientific study. The disorder has been ascribed to many varying factors. In the great majority of cases, however, no physical or structural causes may be found. These forms are spoken of as genuine, idiopathic or essential epilepsy. It is to the psychological nature of these cases especially that the interpretation in this short thesis is to be applied. Thus we have under consideration an apparently sound individual having epileptic attacks of apparent idiopathic or unknown origin, and whose essential epilepsy, therefore, may be considered in the nature of a life-reaction, comparable to a state of rage or anger as seen in bad-tempered individuals, or excessive emotionalism in the supersensitive.

It would appear that a full recognition of an epileptic constitution as such, independent of the deteriorating influence of excessive attacks, which only brings it more markedly in view, was not much recognized before 1860 or 1870. Falret, the elder, an observer of about that time, and Billod (1882), a pupil of Falret, as well as Kirchhoff, who wrote as late as 1890, believed that the peculiar mental make-up of so-called sane epileptics did not possess characteristics distinguishable from that of others in their particular station in life. It is interesting to note the view which a practical neurologist would have upon the presence of a particular and distinctive constitutional make-up, the essential contention of our thesis. Oppenheim, a neurologist of this type, states that the intellect of an epileptic may be "absolutely intact." He cites as examples Cæsar and Napoleon, but quickly adds that we see no such cases to-day. In another division of his work he states that sane epileptics are "often excitable, suspicious and irascible," but hastens to offer an apology for these peculiarities, based upon the nature of the disorder from which they suffer. He finally adds, however, that a normal intellect becomes impaired by the fits.

As soon as the disorders of affect were recognized in psychiatry, independent of purely intellectual ones, a notation of the former began to appear in the literature of the psychology of epilepsy. Now and then one finds in the neurological literature, in contradiction to the psychiatric or psychologic view, an attempt to find an organic basis for the epileptic make-up, just as some authors strive to see many of the mental characteristics produced by the social restrictions which the interned epileptic endures as a result of his malady. Cramer (1912), while firm in the belief that epilepsy proceeds from cortical alterations, becomes confounded and unable to explain the constitutional make-up by such changes. Among the phenomena of latent epilepsy, Hartmann and di Gaspero (1914) believe that, long before the epileptic symptom complex, with its first undoubted paroxysmal phenomena, becomes manifest, an individual may show prodromal symptoms, the relationship of which, however, to epilepsy cannot be maintained with certainty. They cite many variations in affective life from the family histories of epileptics, and find they are hardly ever missing from such histories. Here belong the tendencies to abnormal, endogenous changes of temper, anxious fears and sudden depressions. Apart from the sequelæ and prodromata of an attack, the authors mention great exhaustibility, restlessness, anomalies of disposition, increased irritability, distrust, depression, anxious fears, tendency to violent impulses. In addition to the usual slight mental defects, the authors note a tendency to unreal ideas. There are also memory falsifications. The affective life as a rule shows great departures. There are transitory phobias, ecstasies, and, most common of all, irritable and depressive states. As many as 78 per cent. are said to suffer from these endogenous anomalies of disposition. As a rule consciousness is clear. Under the progressive changes seen in advanced cases of epilepsy, the authors refer to the deterioration. As a rule the subjects react abnormally to external impressions. The affective changes constitute that ethical depravity of the entire personality known as the epileptic character. We see here a morbidly augmented irritability of the temper, with a special tendency to outbreaks of anger. An egocentric contraction of the entire emotional life, pathological lying, &c., may develop.

Féré (1890) believed that the character and manner of epileptics can easily cause suspicion of the disease long before the convulsions appear. The emotional make-up is mobile and explosive; the former rests upon a foundation of "impotence and sadness."

Anyone acquainted with the well-formed peculiar mental characteristics of the frank epileptic in institutional life will easily recognize the classic description of the epileptic make-up as given by Pilcz (1904). In relating anything, the epileptic is never diverted and always arrives at a conclusion. He repeats the same story in the same language, and makes free use of commonplaces, proverbs and biblical texts. As a rule they are pietists, observe religious duties scrupulously. Their devotion is at times exaggeratedly sweet. They flatter by frequently prefixing "good" and "kind" in addressing others. In striking and characteristic contrast to the foregoing is the unusually marked irritability, sensitiveness and egotism. Hübner (1914) also mentions the inclination to piety on the one hand, which plays a great rôle in the thoughts of the epileptic, and on the other the strongly marked egotism which permits him to think only of himself and never of the rights of others.

In accord with the main tenets of the views given in the foregoing citations, one might quote a number of other writers such as Esquirol, Bischoff, Clouston, Ball, Russell Reynolds, Magnan, Howden, Romberg, and others. However, I think it may be proved, so far as opinions and observations of the chief investigators of our subject are concerned: (1) There is a more or less constant affective disturbance in all epileptics, sane as well as insane; that such defect is due to an inherent make-up of the psyche in which mainly an egocentricity and a highly sensitized feeling are given to the individual; and that from this constitutional make-up or alteration the ultimate deterioration of the psyche, intellectually as well as emotionally, is gradually developed, step by step, and if the state is not corrected that this finally and logically ends in real mental deterioration. (2) The epileptic alteration is seen to proceed from the mental make-up or constitution of the individual epileptic long before his malady reaches the convulsive stage, and that the one is but a further and final unfolding of the former.

A few years ago I undertook a study of the character make-up in a small series of essential epileptics *before* they had their first attack, and found these potential epileptic individuals had nothing wanting in the complete picture of the character as seen in those suffering from a long enduring and severe epilepsy, though the character faults were not so glaring as in the frank epileptic; there was also a wide latitude in the quality and amount of defect encountered. It is interesting to compare the attitude of trained clinicians in epileptic colonies and physicians in private practice toward the epileptic individual. The former live more or less in intimate contact with their charges; they recognize their peculiar type—one scarcely detected by the outside physician. Hence, they base treatment largely upon the defects and capabilities of their charges, and grow less and less to depend upon the seizures as such, or to lay stress upon a pure drug or special organotherapy, a practice so common with the general practitioner; yet the position of the trained epileptologist, until recently, has been assumed by many to mean that he is unacquainted with the newer and ephemeral panaceas for this great disorder, and has arrived at therapeutic nihilism. The fact is, the colony physician has a broader view of the whole problem. The physician in private practice sees little of his patient's personality and individual reactions, and often only obtains a record of attacks and their allied symptoms upon which he may base his therapy. If the institutional physician received his patients in an earlier stage, he could probably considerably alter the pessimistic prognosis of epilepsy which now obtains. At present he receives not only the most badly deteriorated patients, but often those so fearfully handicapped by drug and surgical therapy that it takes months to rid the epileptic from these well meant, but misdirected, remedies, so called. It is to be regretted that the majority of text-books continue to state that sedatives are the chief, if not the sole, therapy for essential epilepsy.

II.

Inasmuch as the life reactions of the epileptic character are the distinguishing factors of the make-up, we may sketch the defects of maladjustment at the several epochs of life stress.

At birth the potential epileptic child frequently has periods of meaningless crying. This extra-irritability and sensitiveness is so pathological that it rarely fails of detection and record. For instance, one history states the child "was considered a nervous and irritable child from the day of its birth." A second "fretted continually at the contact of rough clothing," while a third "cried continuously the first three months," although nothing physically abnormal could be detected. One infant nearly went into spasms at the sound of an air-brake. Still another had to be rocked continuously, and slept only during this care.

The next important sequence in such a character is its non-pliability in being taught nursery ethics—that is, obedience and proper daily deportment in the home. These behaviour defects are usually independent of purely intellectual and physical ones, and these infants sit up, creep, talk, and walk at the usual ages. Indeed, in not a few the physical and the intellectual development seem accelerated, and certainly are hyperactive. Extreme lability of mood is a frequent factor. One moment contented, and the next irritable beyond power of appeasement, is often noted. The consecutiveness of purpose in play and capacity to be amused is short, requiring the constant attention of the parent and varying appeals to the child's interest. Tantrum episodes even under the mildest discipline are most common in these difficult children. Their maladaptive defects are soon shown in their association with other children. They demand the play to be arranged to suit them; a game must be played a certain way; it must be continued or stopped as they direct. Their likes and dislikes are extreme. In the school under an impartial discipline these potential epileptics show the most obvious traits. The mood is inconstant, the interest and attention continually vary, and hence their more purely intellectual processes often show wide dissimilarities in evolution. Extremely brilliant in some subjects and quite ignorant in others is the general rule. Because of their extreme nervousness and inability to conform to school routine, many children fail physically and mentally, and cannot take on the normal school training. Some are privately tutored; others are exempted from school discipline and routine except for short periods. Such scholars often appear pale and haggard, the pupils of the eyes are dilated, and they grow lethargic and sullen, or sit day dreaming and yawn. They grow intolerant of the school, and often rebel at its exacting demands. Thus in infancy and early childhood the instinctive defect of the ego fails of proper sublimation; the adjustment to the social and physical environment is incomplete and unsatisfying; and, thrown back upon itself, in consequence there is a reinflation of self-importance and sensitiveness. Childhood, therefore, is a fruitful period for new and unbearable stresses, and, in consequence, epileptic attacks often occur in one predisposed.

The next stressful period is at puberty; here the dissolution of the oftentimes irksome home ties may release some from the galling exactions of home discipline, but most frequently, as in the departure for school with its exactions in deportment, we find the puberty adjustment to work and social demands increasingly onerous. The potential epileptic is not willing to take on a proper attitude of apprenticeship. He has the innate instability of the constitutional inferior, but the good-natured indifference of the latter is not his attitude; his feelings are easily hurt, he has ill-defined paranoid persecutions which cause him frequently to react with violence, insolence, and hatred. He has attacks of rages, sullen moods, and dispirited acquiescence in plans and undertakings. The lack of good-fellowship renders him incapable of co-operative teamwork. The potential epileptic admires co-operation and the doing of big things in the abstract, but the requirements of interdependence and subordination to the main purpose galls and irritates him beyond endurance. During adolescence he begins already to present an odd mixture of primary defects of instincts, together with a commencing deterioration of the higher capabilities of social adjustments known as habit deterioration. In part the latter is protective; that is, under the extreme stresses of this period he may ease off the stress by lowering or evading the exactions of precise behaviour and deportment. Thus, instead of presenting fits for a time, he may take on various types of dissipations. With no real or intimate friends, the potential epileptic holds himself aloof from the demands of the common sacrifice of self and the mutual dependence of social custom. He is a free lance, able and usually anxious to work his own will upon the world. If gifted with extraordinary intellectual endowments, he may succeed for a time and make satisfactory progress towards his goal, but just short of it his egotism and ambition are often fired to new and impossible ends; when the whole scheme seems to him within final consummation, some small trick of fortune adds the last burden of stress upon his mental and physical health, and the individual breaks into frank manifestations of his disorder.

Freed in greater part from the trammels of social concern and demand, the intellectual efforts of the potential epileptic work with less stress than the normal, but in the final and more advanced consummation of his task the social customs of the family, friends and society itself fail to add their small but modifying influence as a directing force to the effort. In consequence new and unforeseen hindrances are added which in turn reduce the mental and physical invulnerability to the minimum, and at such defective periods some physical stress which at a more favourable time would be quite negligible appears as the precipitator of the frank disorder. Thus one learns to discount the common precipitating cause of the epileptic attack; such obvious studies are but surface ploughing of the etiological field in epilepsy. One is apt to think that inasmuch as epilepsy is essentially a disease of early life the great majority are unmarried, because the state in itself bars marriage, but in the vast majority the reason is by no means so simple. It is really because the epileptic is rarely equipped in essential character make-up for marriage. Emotionally and sexually he rarely develops beyond the level of puberty and fails in capacity to attain adult love. Naturally this is to be expected in that the latter demands self-subordination and sacrifice, and above all a tenderness of feeling which is conspicuously lacking in the potential epileptic. In view of the foregoing it is evident that marriage increases demands socially as well as economically and makes not a few potential epileptics break out in attacks. At the threshold of life the vast majority break before or just at this period, which is the point of maximum stress. Almost all potential epileptics, long before their disorder becomes at all acutely manifest, show increasing slowness and a diminished capacity for sustained employment. They show extra fatigue and diminished interest, a partly protective mechanism. A tenacious, consistent, all-around emotional development in a life-work seems impossible to the vast majority. They work fairly well for a time, with plenty of emotional appeal, lavish praise and constant change, all essentially infantile traits of character, but in the end fail to do a thoroughly competent life-work.

III.

The muscular convulsion may be explained hypothetically. We know the convulsive part of the fit in its severer and cruder aspects is comparable to the impulsive movements of the infant. The impulsive fœtal movements begin about the twelfth week of gestation, hence the brain cannot be involved *per se* in their genesis; further, it is known that brainless embryos possess impulsions. There is a short period just before birth in which the amniotic fluid and the uterine wall greatly inhibit the free play of these impulsive movements, but they begin again with renewed activity in the newly born and are slowly inhibited by voluntary control at the end of the nursing period. We do not know just how the impulsive movements are incited further than to surmise that, being of the first, simplest and ontogenetic type of activities of the developing organism, their incitor is from motor centres of the lower order.* In these latter structures are stored up a certain quantity of potential energy which is transformed into actual energy by the blood and lymph stream. With the increasing tissue growth and tension engendered thereby this energy finds its outlet in the random movements of the fœtus and the infant, and their exaggerated distorted presence is seen in the major convulsion of epileptics. Space prevents us from outlining more in detail the essential distinguishing characteristics of the impulsive from the instinctive, reflex and conscious or ideational movements of the infant; this has been done most carefully by Preyer and later correlated into a recent study by Canestrini. Suffice it to say the newer studies on the meaning of the convulsive part of the epileptic fit make careful analysis of all the impulsive movements of the nursling doubly necessary. It will then be found desirable to note their exact relationship in reference to the psycho-sexual development and its defects as shown in the infantilism of the epileptic.

As might be expected the number of the impulsive movements is not great. They may be schematized as those of stretching and bending the arms and legs in the newly born. The movements are sometimes so quick as to resemble the clonic convulsions of a fit. They may be slow, then fast and finally end in clonic movements. Even in healthy infants they may be so slow as to resemble the tetanoid spasm of a beginning focal seizure. Preyer speaks of the muscles involved in the impulsive acts as possessing such a slow, crawling movement that the acts present a striking resemblance to the extension and flexion of the limbs of animals waking from their winter sleep. Such animals, like sleeping children, seen even in the first half of the second year, make genuine fœtal movements which often look as though they were directed against some invisible resistance. This all suggests many of the striking impressions one gains in observing the convulsions of epileptics. Convulsive motions in the infantile impulsions are, however, not generally so frequent in sleep as slow contractions. The latter are frequently attended by spreading and bending of the fingers, which in turn become the rarer toward the end of the second year in all children of sound nervous systems. All these impulsive movements, in the hands especially, are asymmetrical in outline. What are some of the depressors and incitors of these impulsive movements? Deep sleep reduces them to the minimum. Satiation by food greatly curtails them. On the other hand, a duplication of the intra-uterine state by the use of the warm bath encourages them. The movements are then usually slow and rather rhythmic and graceful. One may even see in them the beginning of an expression of pleasure. The face may join in the picture of contentment with slow asymmetric contortions, which semblance has an odd mixture of pleasure with more than a hint of displeasure. The greater part of the impulsions, however, are purposeless, senseless and asymmetric and are found over the entire body from the first day of birth. Writhing and twisting of the body are also frequent accompaniments to the movements of the face and extremities. Just as the infant sinks into deep sleep these impulsive movements slow down and the body usually comes to a state of rest in the fœtal position. The fœtal posture in the legs is kept up longer in advancing child life than that of the head and upper extremities. Many writers have called attention to the fact that no one could consciously duplicate these acts. Then, too, one is strikingly impressed that the infant and the epileptic alike are little fatigued by these most intense and persistent impulsions, which speaks strongly for the unconscious motivation in both their activities. Probably in both subjects the fund of reserve energy being so limited in scope is greater than that of the normal adult as ordinarily expressed in his daily activities. Biologically speaking, we know that the essential vital energy of an individual is probably at its maximum at birth.

We are justified in considering the essential nucleus of the epileptic fit an infantile unconscious striving of displeasure-pleasure pursuit ending in the final goal of a return to infancy, attended by a loss of consciousness and a convulsion; that the convulsion is made up of and flows out of the general striving of the fœtal and infantile tissues as expressed through the lower spinal centres in inducing simple and crude combinations of impulsive movements; that a study of the degree of development of unconscious infantile strivings in the emotional instincts, the desire for an infantile state of omniscience, are paralleled by the kind and character of impulsive movements found in this infantile period of neuromuscular development. Therefore the two main settings in the epileptic fit, unconsciousness and convulsion, are psychical and physical correlates; lastly, that epilepsy in its essential pathogenesis is an error or arrest in this fundamental elaboration or development of the emotional life.

Having schematized the dynamic mechanism of the epileptic fit, we may note that the essential pathogenesis of the disorder as a whole is still to be attacked. Whether the latter rests upon an inheritance of certain psychic traits alone or whether there are certain somatic structural anomalies which do not permit proper emotional and instinctive development into normal adult life one cannot say. I believe such studies, however, narrow the gap between such causes and their psycho-physical expression in epilepsy; and finally, such observations must be of greatest aid in classifying the recoverable epileptics from the irrecoverable ones. It also points the way by which may advance our therapeutics of the disease along the broadest biological lines of educational and moral treatment. In this connexion one may note that MacCurdy has tentatively formulated the idea that the sudden loss of consciousness in epileptics liberates a muscular anarchy of "clotted mass of movement" of many different lower levels or physiologically controlled centres in the brain and spinal cord, and that the convulsive part of the fit is a released neuromuscular mechanism or series of mechanisms entirely secondary to the loss of consciousness, the main defect of the epileptic state. He holds the same fundamental postulate as to the psychological meaning of the epileptic fit as we have formulated it.

IV.

Given the inherent defect of make-up, it is easy to comprehend how all forms of undue physical and mental stress may operate deleteriously upon the epileptic. The gradations of epileptic reactions vary from day-dreaming, lethargies, petulance, sullenness and outbursts of impatience, until there succeeds a series of minor attacks or a severe major convulsion, when the lowering skies in the epileptic's life are dispelled for a time until the stresses again accumulate to an explosive level. So long as normal consciousness is maintained the stress may work its evil consequence in ways well known to all. The epileptic reaction from its mildest to its severest manifestation is really a protection, for it obliterates reality and reduces the subject to the lowest level of organic response, that of a comatose state. Hence the fit is really a protective mechanism, psychologically considered at least. It withdraws or reduces the subject's attachment and adjustments to reality. It dispels an intolerable demand and the epileptic retreats to a state of harmony and peace. In the retreat after incomplete attacks we frequently encounter unconscious strivings and conflicts that have baffled the subject. So exogenous causes, physical and mental in character, slight as they may seem to be, precipitate a conflict which fires the gun, and a series of conflicts of different levels in the unconscious are exposed until after the severe major attack the subject is reduced to the lowest level, comparable to earliest infantile life. When this hypothesis was enunciated a few years ago it was as yet uncorroborated by exact data, but since that time innumerable studies bearing out every contention of the mechanism have been deduced. Thus we find in the mild and transitory deliria of the automatic phase after petit mal attacks the subject may say or do certain things which may be pieced together and minutely analysed. Like the mental content in maniacal states or drug and fever deliria, these spontaneous productions have to do with the conflicts of everyday life. Then appear the successive deeper levels of emotional strivings and conflicts. For instance, at first the epileptic attempts to rid himself of an onerous task or unpleasant companions; sexual strivings are uncovered and in the deepest level he has made a retreat to the home, is in the cradle or the mother's arms, &c.

The view here expressed in reference to the psychology of epilepsy is not essentially new either in principle or application of treatment. It but gives us a more rational understanding of the nature and mechanism of the disorder itself and renders the plan of handling and treating the essential disorder more understandable and practicable. One can easily comprehend that the point of attack in the treatment of such a disorder rests upon an effort toward training out the constitutional defects of personality and teaching these patients to make a better adjustment to life.

We therefore must conclude that essential epilepsy as regards causation is dependent upon a primary congenital defect or inheritable defective instincts of natural and healthful adaptations to reality, the epileptic make-up or constitution, and that at successive periods in life, when emotional and physical stresses of an intensive character are encountered, the individual so constituted has epileptic reactions such as fits, disturbances of temper, lethargies, and various psychic phenomena. The fit is essentially a regressive phenomenon, a flight from undue stress into unconsciousness, the deeper manifestations of which, the convulsive phenomena, are superadded. By analysis of the states of automatism of epilepsy, by the conscious analysis and the dream states, one gets to know the depth of regression and the infantility or crudity of the essential instincts of the individual epileptic. In consequence the future lines of treatment heretofore empirically undertaken by reducing the life stresses is now made much more patent by our exposition of the instinctive defects: that the proper treatment of epilepsy is an intensive and persistent educational training in its broadest and deepest sense, together with the correction of such mental and physical disorders as are remediable by more conscious methods, as occupation, educative play and healthful interests and pursuits.

REFERENCES TO FULLER STUDIES BY THE AUTHOR OF THIS PAPER.

"Nature and Pathogenesis of Epilepsy," *New York Med. Journ.*, 1915, vol. ci, pp. 385, 442, 515, 567, 623.
"A Personality Study of the Epileptic Constitution," *Am. Journ. Med. Sci.*, 1914, vol. cxlviii, p. 729.
"Clinical Studies in Epilepsy," *Psych. Bull.*, *N.Y.*, 1917, vol. xi, p. 21.
"The Psychological and Therapeutic Value of Studying Mental Content during and following Epileptic Attacks," *New York Med. Journ.*, 1917, vol. cvi, p 677.
"A Further Study of Mental Content in Epilepsy," *Psych. Bull.*, *N.Y.*, 1917, vol. xi, p. 483.
"The True Epileptic," *New York Med. Journ.*, 1918, vol. cvii, p. 817.
"A Study of Certain Aspects of Epilepsy compared with the Emotional Life and Impulsive Movements of the Infant," *Interstate Med. Journ.*, 1915, vol. xxii, p. 969.
"Treatment of the Epileptic, based on a Study of the Fundamental Make-up," *Journ. Amer. Med. Assoc.*, 1918, vol. lxx, p. 357.

THE PSYCHOGALVANIC REFLEX: A REVIEW.

BY E. PRIDEAUX.

(*From work carried out at Cambridge for the Medical Research Committee.*)

WALLER [45], during his investigations on the electrotonic changes of excitability of the motor and sensory nerves of man in 1880, noticed irregular galvanometric deflections, which at that time he could only explain as being due to slight alterations of contact between the skin and the electrodes caused by muscular movement. Féré [7] was the first to point out in 1888 that an emotional reaction was accompanied by electrical changes in the human body, demonstrable by galvanometric deflections, when the resistance of the body to a weak electric current was being investigated; he noted that these changes were correlated with the alterations in volume of the limbs as shown by the plethysmograph, but reported that the galvanometric deflections had been insignificant in the normal persons he had experimented upon, and were only established for hysterical patients. In Féré's opinion the changes were due to a lowering of the resistance of the body under the influence of emotional states.

In the same year, independently of Féré, Tarchanoff [42], working on the electromotive changes in the body, and using no external current, showed that the phenomenon was a normal one, and occurred in all healthy individuals, when two non-polarizable electrodes attached to asymmetrical parts of the body were connected up with a galvanometer. Tarchanoff concluded that "the galvanic phenomena observed in the skin, which are concomitant with different kinds of nervous activity, should be considered as due to an active state of the sweat glands, which at this time produce a secretory current."

The work of Féré and Tarchanoff remained for several years unnoticed, and it was not until E. K. Müller [24], an electrical engineer without any knowledge of the previous work, rediscovered Féré's phenomenon in 1904, and communicated his results to Veraguth [43], who gave the name "psycho-physical galvanic reflex" to the phenomenon, that any methodical research was carried out. Although Veraguth's term, now shortened to "psychogalvanic reflex," has come to be used indiscriminately for all electrical changes shown by the galvanometer due to psychophysiological processes, it is necessary, in order to avoid the confusion in which some investigators of this subject have entangled themselves, to deal separately with the phenomena as reported by the original observers: (1) Galvanometric deflections due to production of some electromotive force when no external current is used (phenomenon of Tarchanoff); and (2) Galvanometric deflections due to apparent change of resistance when a weak electric current is passing through the body (phenomenon of Féré).

APPARATUS EMPLOYED TO DEMONSTRATE THE REFLEX.

(1) *When no external current is used.*—Tarchanoff [42] employed non-polarizable clay electrodes attached to the skin by means of hygroscopic pads saturated with saline solution. These were connected with a Meissner and Meyerstein galvanometer. The electrodes were applied at different times to asymmetrical parts of the body such as the hands and fingers, feet and toes, face and back, whilst the subject was kept at complete rest. Experimenting in this way he found that psychic stimuli, real and imaginary, provoked galvanometric deflections. Later, investigators have followed this method with but slight modifications. Sticker [41] repeated Tarchanoff's experiments in 1897, and Sommer [39] in 1902. Fürstenau [8] experimented on the physical nature of the phenomena and the effects of different electrodes. Sidis and Nelson [38] relied chiefly on platinum hypodermic electrodes, used a d'Arsonval galvanometer ("sensibility 225 megohms"), and took photographic records of the deflections; they showed that even from symmetrical parts of the body, in the case of the two hands, a galvanic reflex was produced. Gregor and Loewe [15] and Philippson and Menzerath [30] employed an Einthoven's galvanometer with photographic recording apparatus, the former relying chiefly on Ostwald's electrodes, and the latter on Einthoven's zinc sulphate electrodes.

Radecki [33], using zinc electrodes, got similar results with Lippmann's capillary electrometer, and found that the electromotive force produced was about 0·005 volt.

(2) *When an external current is used.*—Féré did not give any particulars of his method. The usual technique of most of the investigators is a modification of that employed by Veraguth. He introduced a d'Arsonval mirror galvanometer with a shunt into a circuit, through which a feeble electric current from one or two Leclanché cells was being conducted through the body, the places of entrance and exit of

the current being the palms of the hands, and the electrodes being nickel-plated brass cylinders. He measured the deflections of the galvanometer on a horizontal celluloid scale, on which the light from the mirror registered its movements. A similar technique, with the addition of a device for recording the deflections by hand on a kymograph, was adopted by Peterson and Jung [27, 28], using copper plates as electrodes, Ricksher [34] with brass plates, and Radecki [33] with zinc electrodes. Sidis and Kalmus [37], and also H. Müller [25], used liquid electrodes—glass vessels filled with concentrated saline solution, in which large copper electrodes of about 500 cm. area were permanently placed, and in which the hands or the feet could conveniently rest. Gregor and Loewe [15] were the first to take systematic measurements of the deflections in terms of apparent resistance, and compare it with the resistance of the body by means of a Wheatstone bridge. Waller [47, 49] independently has adopted this method as the most convenient for accurately determining the magnitude of the deflections in terms of ohmic resistance, or, reciprocally, of conductance, introducing also into the circuit a potentiometer delivering one-tenth of the E.M.F. of a Leclanché cell for purposes of calibration, by means of which the deflection can also be directly measured and expressed in terms of voltage; he passes his main current from two Leclanché cells through one hand, and applies the electrodes, usually zinc discs covered by chamois leather moistened with 0·6 per cent. saline solution—one on the dorsum and the other on the palm of the hand. By this method he has shown that the reaction expressed in terms of voltage may be 0·5 volt.

PHENOMENA OF THE REFLEX.

The only fact about which there is general agreement amongst the investigators on this subject is that the phenomena of the reflex have been definitely established, both with and without the use of an external current.

(1) *Galvanometric deflections due to psychical causes.*—Any stimulus giving rise to an emotion, such as the unexpected ringing of a bell, flashing a light, a pin-prick or a burn, after a certain latent period will cause a deflection which is proportional to the subjective state aroused. An actual stimulus is not necessary, the expectation of the stimulus is sufficient, and it is only necessary for the experimenter to walk behind the chair of the subject for a deflection to be produced. The threat of

a stimulus often provokes a greater reaction than the stimulus itself. Also calling up memories of painful experiences, or referring to questions concerning which the subject may have pin-pricks of conscience, will give a deflection.

(2) *Galvanometric deflections due to physical causes.*—Alterations in contact between the skin and the electrodes will cause deflections, but these are easily recognized. When using liquid electrodes, this deflection is always in an opposite direction, due to withdrawal of the hands or feet from the solution: when using metal electrodes, firm pressure on them can produce a very slight deflection in the direction of the emotional reaction, but muscular movement causing diminution of contact will produce a deflection in an opposite direction. These deflections can be distinguished by the fact that they take place immediately, and are not characterized by a long latent period.

(3) *Galvanometric deflections due to physiological causes.*—A deep inspiration, cough, yawn or sigh will produce a large deflection, whilst ordinary respiratory movements will not visibly affect the galvanometer. These deflections have a latent period of the same order as the emotive deflections.

(4) *Latent period.*—The latent period varies in different people, and in the same people at different times. It is of an average duration of two to three seconds. The periods recorded by different observers vary slightly, as some—Veraguth [43], Knauer [19]—make it longer, having measured the time from the commencement of the stimulus to the summit of the reaction, instead of to the commencement of the reaction, as is now generally done.

(5) *After-discharge effect.*—The reaction provoked by the excitation persists for some time. This time varies considerably in different persons; it may last only ten to twenty seconds, or it may persist for a few minutes.

(6) *Fatigue of reflex.*—After a stimulus has been repeated a few times it begins to lose its emotive effect, and very little reaction then occurs. It cannot be regarded as a true reflex fatigue. After a subject has been experimented upon for some time and has become familiar with the procedure, he may give no reactions until some new and unexpected stimulus catches him unawares.

(7) *Diminution of reflex as result of fatigue.*—The same subject will react differently to the same stimuli at different times of the day, and at the same times on different days, according as he feels in good or bad health. Physical or psychical fatigue diminishes the reflex.

possible to produce a contrary electromotive force due to polarization effects of two volts in the frog and of six volts in man.

With an alternating current the phenomena due to the polarization effects are reduced to a minimum. It has recently been shown by Gildemeister [12] that the resistance of the body varies according to the method of measurement. With the constant current the resistance depends on the duration and intensity of the current; with alternating currents the resistance is smaller the greater the frequency. After the removal of the skin the variations are smaller, whilst observations on the skin alone show the fluctuations just as the intact body does. It was pointed out by Aebly [2], working under Zangger, that the resistance of the body by Kohlrausch's method remains the same whether or not a current was passing through it, and that the resistance of the body to the alternating current does not alter under the influence of emotional reaction. By analogous experiments Gildemeister [9, 11] demonstrated the presence of the psychogalvanic reflex without any alteration in ohmic resistance, and Schwartz [35] got identical results in the frog.

It is now obvious why a diminution in the strength of the current reduces, and may if sufficient practically abolish the large reaction usually obtained when using a current of about 2 volts, for alterations of the current will affect the polarization of the skin.

We can, therefore, definitely exclude bodily resistance as a cause, and point to the polarization of the skin as the chief factor in the production of the reflex.

Knauer [19], in his criticism of Veraguth's book, "Das psychogalvanische Reflex-phänomen," got very near to this view, but it was H. Müller [25], and Aebly [2], who first drew attention to the phenomena of polarization and recognized that the reflex was a polarization effect; they held that the increase in conductivity of the body was due to a diminution of the electromotive force of polarization, and this view was upheld by Piéron [31], and in part by Gregor and Loewe [15]. The polarization effect then might be due to a diminution of the electromotive forces of polarization, as held by Müller, Aebly, and Piéron, or to a diminution of the resistance set up by the membrane where polarization occurs. Waller [48] has settled this point by the experiment here quoted: "If a potentiometer is put in series with the subject in the fourth arm of the bridge, we shall obtain indications that will enable us to decide between the two alternatives—electromotive force increased or resistance decreased. If the former, a known E.M.F. from the potentiometer should give the same deflection before and during an emotive change. If the latter, the potentiometer deflection should be increased during the emotive change. And in point of fact this second alternative is what actually occurs; whenever I have measured the resistance in ohms before and during emotive excitement, I have found that the potentiometer deflection was inversely proportional with the resistance." We must conclude, therefore, that the psychogalvanic reflex in the presence of an external current is brought about by a diminution of the resistance of the skin due to the effect of polarization, and that this polarization is influenced by an emotional reaction.

PHYSIOLOGICAL PROCESSES CONCERNED IN THE CAUSATION OF THE REFLEX.

The physiological processes that will have to be considered as being possibly concerned in the causation of the reflex, both as causes for the change of potential between the electrodes when using no current, and as causes for the changes in the polarization of the skin in the presence of a current, are: (1) Unconscious muscular movement; (2) changes in the capillary circulation; (3) respiratory changes; (4) secretory activity of the skin; (5) nervous excitation through the intermediary of the cerebrospinal or automatic nervous system; (6) biochemical changes in the skin, or to a combination of these. But slight changes sufficient to cause a difference of potential, such as currents of action due to unconscious muscular movement or secretory activity, which may be the concomitants of emotion, are not sufficient to account for the large changes obtained when a current is being used.

(*a*) *Reflex due to production of an electromotive force.*—Investigators who have given theories to account for the changes of potential are Tarchanoff [42], Sticker [41], Sommer [39], Sidis and Nelson [38], Radecki [33], and Philippson and Menzerath [30].

Tarchanoff relied entirely on the activity of the sweat glands to account for the phenomenon, and explained the galvanometric deflection as being due to a secretory current. He showed that by placing non-polarizable electrodes on different parts of the body the area most rich in sweat glands became negative to the less glandular area. This question of the activity of the sweat glands must be taken up later when dealing with the processes concerned in the presence of a current.

Sticker repeated Tarchanoff's experiments, and, whilst not opposing his secretion theory, held that the changes in capillary circulation also played an important part; he based his view on the work done by

Hallion and Comte [16] on the influence of emotion on capillary circulation. Radecki's experiments support Sticker's theory; his conclusions are that the reactions are due to changes in the circulatory and secretory systems; that the changes of potential result from discharge of electricity which takes place during glandular activity, and that these changes depend directly on secretory and excretory processes, and indirectly on the capillary circulation. Though it cannot be denied that the above processes do cause changes of potential, there are serious objections to considering any of them as the specific cause of the psychogalvanic phenomenon. As Sidis [37] has shown, when using no current, exclusion of the skin by the use of hypodermic needle electrodes, or covering the skin with shellac and paraffin, leaving only the nails exposed, does not prevent the occurrence of a galvanometric deflection. Also exclusion of the circulation, in man by means of an Esmarch's bandage and in the frog by ligation of the arteries supplying the limb, does not materially affect the reaction. Sidis and Nelson [38], on the other hand, having excluded the skin and circulation, claim that the phenomenon is exclusively muscular in origin. They have recorded some very careful experiments from this point of view, and showed the presence of galvanometric deflections due to muscular contraction when the other causes were excluded. But the difficulty in accepting Sidis's conclusion is that these experiments were done on animals; that they do not mention anything at all about the latent periods, nor is it possible to deduce them from their records, and that they did not try any experiments in man from the point of view of excluding muscular contraction and leaving the skin as a possible factor. They relied on the fact that the deflection was abolished in the curarized frog. It is possible that they were in reality investigating the currents of action due to muscular contraction, and were only repeating the classical experiments of Du Bois Reymond [6]. The most recent work has been done by Philippson and Menzerath [30], who conclude as the result of their experiments that the origin of the difference of potential causing the galvanometric deflections during emotional excitement cannot be attributed to any one specific cause. They hold that it is due to the following combination of causes: (1) Exclusively cutaneous by changes in the activity of the sweat glands or in the chemical products secreted; (2) musculocutaneous when the right hand becomes positive to the left, which phenomenon they explain after Du Bois Reymond [6] as being due to the distension of the skin following involuntary contraction of the right hand; (3) exclusively muscular. They also confirmed Tarchanoff's [42] statement that there is no correlation between the reflex and respiration, for the pneumogramme and the electrogramme do not show any parallel. It would seem *a priori* that the same changes in the skin which will account for the reflex in the presence of a current would also set up a difference of potential and produce the electromotive force reflex. The evidence to be put forward as the result of experiments with an external current tend to prove that none of these causes can be regarded as the exclusive cause of the reflex, and at present the problem is still unsolved.

(*b*) *Reflex due to change of resistance.*—Muscular movement can be definitely excluded by the experiments already cited.

The effects of circulation as a specific cause may also be excluded, for the reaction is not appreciably affected by the application of an Esmarch's bandage, as originally shown by Veraguth [43]. In spite of these experiments, Radecki [33] still maintains this view. He holds that the reflex is due to a vasodilatation provoked by a psychic excitation, and that the changes in the circulation cause an increase in gaseous exchange in the organism and thereby increase the conductivity. It is true that vaso-dilatation may cause a reaction in the same way that a forced inspiration will, but it is not the cause of the galvanic reflex.

The influence of the respiration deserves more attention than has yet been given to it. A deep inspiration, a cough or a yawn produce a most marked effect, often greater than that produced by emotion, whilst ordinary respiratory movements do not affect it. At first sight it would seem that here we might have an explanation of the phenomenon; but in an extensive study on this question, Peterson and Jung [28] have shown that there is no constant relation between galvanometric and pneumographic curves. They conclude: "When the emotions are very labile and show the most marked excursions in the galvanometer curve, the respiratory curve is often regular and even. On the other hand, where the galvanometer curve is marked by little fluctuation, or even by none, there will often be most decided variations in the pneumographic curve." These authors, referring to the rise in the galvanometric wave due to inspiration and coughing, state: "We are inclined to think that this rise may also be psychic, that is emotional. Certainly, in the curve we observe exhaustion by repetition of the command to cough or breathe deeply, as in the case of other analogous stimuli." It would seem more likely that the exhaustion in these experiments, which is, however, slight after fifteen deep inspirations and fourteen coughs, is due to physical fatigue, and that the change in polarization of the skin produced by a deep inspiration is a purely physiological effect. If this is the case, then the changes in the skin due to a deep inspiration or coughing are analogous with the changes produced by emotion, but they are independent of each other. However, a study of the biochemical changes in the skin due to forced respiration might possibly give us the key to the psychogalvanic phenomena.

The theory which has been most strongly supported is that the phenomenon is due to alterations in secretory activity. Veraguth [43] supposed that the seat of the change of conductivity was the skin, and thought that this conductivity changed, for the following reasons: "The cells of the body, like colloids surrounded with a membrane, ought to show, each in its own particular way, a specific obstacle to migration of the ions. This way varies in the same cells, both according to general tone and according to the alterations in their state brought about by excitation." He excludes participation of the perspiration itself, for the results were similar in hands made dry with formalin, but he does not exclude secretory activity. He quotes one case in which he placed a belladonna plaster on his subject for three hours, then took it off and rubbed the hand with liquid extract of belladonna, and found that the psychogalvanic reflex was diminished.

Peterson and Jung [28] attribute the phenomenon to a secretory activity. "It would seem that the sweat glandular system is the chief factor in the production of this electric phenomenon, inducing on the one hand under the influence of nervous irritation a measurable current and on the other hand altering the conductivity of the current. Change in resistance is brought about either by saturation of the epidermis with sweat or by simple filling of the sweat gland canals, or perhaps also by intracellular stimulation; all of the factors may be associated." This view has been very generally accepted. Gildemeister [9], and more recently Philippson and Menzerath [30], have accepted this explanation, relying apparently on the experiments of Leva [21].

Leva found that in ten subjects a subcutaneous injection of 1 mgrm. of atropine sulphate entirely abolished the reflex; that directly after the injection, and for the next ten to fifteen minutes, the galvanometer deflection was of the usual order, but that after fifteen to twenty-five minutes it diminished rapidly, and after about thirty minutes even the strongest stimulus gave no reflex. The results of Waller's experiments from this point of view [50] are directly opposed to those of Leva. Waller states: "I have taken great pains to satisfy myself about the action of atropine upon the emotive response, and have failed to find any evidence that it possesses any action at all upon the response." This view is supported by the experiments of Markbreiter, working in collaboration with Waller [22]. In one of these experiments, even after an injection of 2 mgrm. of atropine sulphate into the palm of one hand, when the usual pharmacological action was marked, no effect was produced on the galvanic reflex, and equal responses of the usual order were obtained from both hands. The inconsistency in the results of Veraguth, Leva and Waller seems difficult to explain. Neither Veraguth nor Leva give any photographic records of their experiments, nor any detailed accounts of their technique. Certainly in Veraguth's experiments the electrophysiological conditions did not remain constant during the test. It is possible that Leva, who used non-polarizable electrodes, was only dealing with a deflection due to a small electromotive force, caused by a current of action due to secretion, the nature of which was determined by Hermann [17] in his experiments on the forearm. In this case Leva was only repeating experiments analogous with that of Hermann [18], who showed that atropine suppressed the ascending current due to secretory activity, which was normally produced by excitation of the sciatic nerve in the curarized cat.

Further confirmation of Waller's atropine experiments is afforded by his results with pilocarpine and the calcium chloride capsule experiments, devised by him [46] for the measurement of insensible perspiration. He has demonstrated [50] that an increased emotion, as shown by the galvanometer, is not necessarily accompanied by an increased secretion, and in fact in the four experiments on the same subject which he quotes the contrary was the case, and an increased emotive deflection was accompanied by a decrease of secretion. Waller concludes: "While it is no doubt true that the deflection is due to diminished resistance, and that perspiration causes diminished resistance, it does not follow logically that the deflection is due to perspiration, since there are many occasions on which the emotive fall of resistance occurs without augmentation of either sensible or insensible perspiration." Waller's evidence is so strong that we must conclude that the change in polarization, and consequently the psychogalvanic reflex, does not depend on the secretory activity of the skin.

There remain therefore only the influence of nervous excitation, through either the cerebrospinal or autonomic nervous system, and changes in the biochemical processes in the skin dependent on them, to account for our reflex. Very little work has been done from these points of view.

V. J. Müller [26], in researches on the *Macacus cynomolgus*, found that the reflex was suppressed by perineural injection of an anæsthetic round the nerves supplying the palmar surfaces of the upper limbs, but that in man the reflex was not suppressed, but only diminished. Section of the nervous trunk gave an immediate suppression, and this suppression lasted while the electrodes were in contact with the palmar surfaces, but at the end of some time the dorsal regions, which did not previously give reactions, then gave them. This is not explainable, and has not been followed up by further work to verify this result. Sticker [41], working only on Tarchanoff's phenomenon, noted that the reaction occurred in both anæsthetic and analgesic skin areas, organic and functional, but there is not sufficient information on this point in regard to Féré's phenomenon. Any further consideration of the physiological nature of the reflex must at the present time be hypothetical. Waller's view is that the emotive phenomena belong to nutritional or trophic changes in the course of metabolism, that "it is a sudden brief intensification of a slowly fluctuating state in nutrition," and imagines "the changes as brought about through what we are accustomed to designate as 'trophic' nerve fibres." He is now investigating the subject from this point of view.

Meanwhile we must conclude (1) that the ultimate nature of the reflex is still an unsolved problem; (2) that muscular, vasomotor, respiratory and secretory changes may be concomitant with emotional reactions, but that none of them is exclusively responsible for the psychogalvanic reflex; (3) that the probable cause is to be found in the biochemical changes in the skin, which are brought about by nervous excitation, but that there is no evidence to show what is the nature of these changes, or through what nervous channel this excitation takes place.

CLINICAL AND PSYCHOLOGICAL STUDY OF THE REFLEX.

The psychological problems involved in a study of the psychogalvanic reflex would seem to open up wide fields for inquiry, and yet on this side little systematic work has been done. Whatever may be the actual nature and causation of the phenomenon, it still remains for the psychologist to determine its significance, and also the conditions under which it varies, and to investigate the mental processes concerned in its excitation. The difficulty in interpreting the results of different observers is, firstly, that they are obtained under varying electrophysiological conditions, and are not directly comparable; and, secondly, that the usual variations obtained between different individuals are not much greater than those obtained in the same individual at different times under different conditions of health; and, thirdly, that no standard measurement under constant electrophysiological conditions has yet been determined by any observer for this amount of variation in the same individual.

Waller [49] has worked out the periodic variations of conductance of the palm of the hand for one subject, and has found that they have a diurnal periodicity, the resistance being greater during the night than during the day. The question as to whether there is any constant relation between the strength of the psychogalvanic reflex and the apparent resistance of the skin as determined at the beginning of the experiment must remain open. Waller [49] thinks there is, but is not yet able to state it with complete numerical evidence. On the other hand, Ricksher and Jung [34] state that there is no relation between the two. If this question were settled in favour of Waller's assumption, then equally valuable evidence from a clinical point of view might be afforded by recording the apparent resistance of the skin, without any observation of the reflex itself. This would be a continuation of the original work of R. Vigoroux [44], who pointed out in 1879 that the resistance of the skin was considerably diminished in cases of exophthalmic goitre, and increased on the anæsthetic side in patients suffering from hysterical hemianæsthesia.

The observations which have been made from the clinical standpoint are mainly on the value of the reflex in the diagnoses of hysterical conditions, particularly hysterical anæsthesia, and also in the differentiation of the psychoses. Veraguth [43] has recorded cases of tabes, syringomyelia, &c., to show that, after painful stimuli in anæsthetic and analgesic skin areas of organic origin, there is no psychogalvanic reaction, when the stimuli are not appreciated subjectively by the patient as such. He has shown the importance of bandaging the patient's eyes and of giving no verbal indication of the stimulus during such experiments. If the patient could see or hear the approach of the stimulus, a normal psychogalvanic reaction was then given after stimuli in the anæsthetic and analgesic areas, due to the psychical effect of expectation. He also records one experiment with a cat, in which the reaction was abolished during ether narcosis, and in which, after dividing the spinal cord in the lumbar region, and allowing the cat to regain consciousness, a painful stimulus to the tail gave no reaction, whilst one to the front paw gave the usual reflex. He showed, on the other hand, that stimuli in anæsthetic skin areas of functional origin always gave the usual reaction, and this has been the experience of all observers. I have also made use of the reflex for the purpose of diagnosing a functional amnesia from an organic one, and in one case in which the patient had a complete loss of memory for all his war experiences, I was able, by means of his galvanic reactions, to trace out roughly the history of the patient's movements during the war, and to determine the place where he was "blown up." In addition, I have used it as the only trustworthy test to decide whether a deafness is a true organic one or not; the amount of functional deafness in organic cases can be determined in this way. If the patient states that he does not hear the stimulus, but at the same time gives a reaction, then that amount of deafness is functional. It is impossible to state at present whether in these cases a negative reaction always indicates an organic deafness, but, by analogy with the cases of organic anæsthesia, it seems probable that this is the case.

Before studying the variation of the reflex in the psychoses, it is necessary to investigate the mental processes concerned in its excitation. So far it has been tacitly assumed that the reflex is simply an indication of an emotional reaction. Though it is easy to show that all emotional stimuli provoke galvanometric deflections, it is not so easy to say whether the deflections provoked by the psychical stimuli may not also be due to the effects of intellectual processes. Radecki [33] concludes that "the phenomena take place in the human organism exclusively as an expression of our emotions and affective states." Binswanger [4] has also expressed a definite opinion on this question to the effect that "among mental activities only the affective processes, in Bleuler's sense, have any influence. Intellectual work or simple sensory impression have no effect." On the other hand, it is the experience of most observers (Peterson and Jung) [28], Veraguth [43], Starch [40], Gregor and Gorn [14], that mental effort does produce a small reaction. Starch [40] concludes that "all the different types of mental processes produced by the various stimuli were accompanied without exception by galvanometric changes. Emotional processes and muscular activity produced the widest deflections, while habitual mental activity and the process of visual attention produced the least deflections. Quiet mental activity, even when involving considerable effort, produced small galvanometric changes. The degree of intensity of emotional experiences corresponds very closely with the amount of deflection." It is impossible to say how much affect accompanies an intellectual process such as an arithmetical calculation, due to the effort of attention. Very often a strong affect is produced by the surprise at the question, or by the embarrassment and possible annoyance that the solution of the problem may be incorrect, and this can only be decided by introspection on the part of the subject. Even if a pure intellectual process does produce a reaction, it is very small, and is in no way comparable with the deflections produced by an affective process, so that for all practical purposes it may be considered that among mental activities only the affective processes have any influence in exciting the reflex. This is also confirmed by the fact that after repetition of the stimulus, thereby reducing its emotive effect, the reflex becomes correspondingly diminished.

The next question, which does not seem to have occupied the attention of any of the observers, is whether the deflections are not equally due to the attempt to suppress the emotion aroused, and to prevent it from operating. As Waller [49] puts it, "the more perfectly an examinee can control the visible signs of the emotion, the more violently is the galvanometer deflected through the palm of the hand by reason of his suppressed emotion." Waller only makes this statement in support of the fact that the palmar electrical sign is not under voluntary control, but it would seem to have a greater significance than he attaches to it. Waller [51] is of the opinion that the phenomenon is not a psychogalvanic reflex, and prefers not to use the prefix "psycho," because in his view the phenomenon has nothing to do with the conscious mind. He states "it is not a cortical phenomenon, it is a subcortical phenomenon." I have been led to think that there is a constant correlation between the visible signs of emotion as shown by muscular movement, which is under voluntary control, and the psychogalvanic reflex, which is not directly under voluntary control. The greater the visible signs of emotion, and the greater the muscular movement, the less is the response on the galvanometer. From this point of view a high psychogalvanic reflex would indicate not merely an emotive response in Waller's sense, but in addition an increased sensitiveness on the part of the subject which may be partly due to experimental conditions and also an increased power of control. Whereas a low reflex would indicate that the subject was of diminished sensibility or had little control over his emotions, it might also mean that the subject was of low emotivity, as Waller says, but it would not necessarily be so. In my view then the phenomenon is as much cortical as subcortical, and is not a true indication of the emotivity of the subject.

Not only has the psychogalvanic reflex been evoked by emotional reactions concomitant with mental causes known to consciousness, but also it has been shown by Morton Prince and Peterson [32], Peterson and Jung [28], Binswanger [4] and others, that stimulation of ideas in a state of repression in the unconscious ("complexes") will also produce a reaction. Morton Prince and Peterson carried out experiments on Sally Beauchamp in her different states of personality, and showed that a memory of an experience which could not be recalled to consciousness provoked the usual reactions; they also showed that the reaction was the same in hysterical anæsthesia as in the normal, and that objects in the peripheral field of vision not consciously perceived were recognized sub-consciously (with Prince "co-consciously"). Radecki [33], working with Abramowski, showed that reactions could still be obtained through the sub-conscious by giving stimuli whilst the attention of the subject was concentrated on some mental problem, when the subject was not able to recall the fact that such stimuli had been given. Peterson and Jung [28], and particularly Binswanger [4] have done elaborate experiments on "association" tests to show that the galvanic phenomenon, like reaction time and alteration of reproductions, may give evidence of the existence of an unconscious "complex." These reactions have been so marked that they have given it as their opinion that reactions are evoked particularly by "complexes," and they have used it extensively as a "complex" indicator. Binswanger points out that an existing affect or lasting concentration of the attention on matters irrelevant to the experiment inhibits the psychical elaboration of the stimulus, and that, therefore, under these conditions the stimulus does not provoke much emotive reaction or give a psychogalvanic reflex. Prolongation of reaction time without a corresponding increase of the galvanic reaction may occur when there are purely intellectual difficulties, such as indistinct perception of the stimulus word, but more frequently under the influence of perseveration. Increase of the psychogalvanic reflex without corresponding prolongation of the reaction time may occur for purely linguistic reasons, when a genuine emotional reaction takes place, but is masked if an easy reaction word is at hand, favouring a habit reaction or sound association. In the latter case the galvanic reaction is the only way of discovering the real significance of the stimulus word. This significance is often not known by the conscious mind and can then only be established with certainty by psycho-analysis. The psychogalvanic reaction is therefore a much more delicate and correct "complex" indicator than the "reaction time" test.

The influence of hypnosis on the reflex has been recorded by Moravcsik [23]. He quotes only one case, but this is a particularly interesting one, from the fact that the reflex was almost completely abolished as the result of suggestion. The reaction to a pinprick, after the suggestion that the hand was hypersensitive, corresponded to 35 mm. on his scale, and after the suggestion that his hand had become anæsthetic the reaction was only 5 mm. After the suggestion that the subject was deaf and blind, auditory and visual stimuli provoked no reactions at all. This result is difficult to explain, and is not in accord with our usual experience of functional anæsthesia and deafness in hysterical cases. Also it is not in accord with the experience of Morton Prince and Peterson [32], already quoted. I got different results in some experiments I did with Waller two years ago, which were not reported, but of which photographic records were taken. These subjects, three men and one girl, had been hypnotized by me previously to the experiments two or three times, and were able to go into the somnambulic state quite easily; one of the men had had three teeth extracted and an operation for whitlow, and the girl had had two teeth stopped, with the extraction of a live nerve from one of them, under hypnosis, without any visible signs of the perception of pain or conscious memory of the operation. Records were taken both in the normal and hypnotic states, and there were no appreciable modifications in the galvanic reactions, either to painful or auditory stimuli. Moravcsik does not record the normal reactions of his subject, and does not state whether the subject was previously acquainted with the experimental conditions of the reflex. It would, for instance, be easy for a subject who had become acquainted with the physiological processes causing a deflection, and who gave normally a small deflection, due to familiarity with the stimuli, to produce a big deflection by taking a deep inspiration. On the other hand, there are undoubtedly some persons who can influence the rate of their pulse, and possibly even the surface temperature of their body by suggestion. Moravcsik [23] has recorded one such case in which the pulse-rate was altered from 84 to 104 and the temperature from 37·3° C. to 38° C. Presumably such subjects would be able to influence indirectly the psychogalvanic reflex. That voluntary effort can in certain cases affect the reflex in the direction of diminution is claimed by Radecki [33], who thinks that this effect is due to the influence on respiration, and also to the fact that voluntary effort produces attention which interferes with the emotional reaction evoked. The most probable explanation would seem to be that the attention necessitated by voluntary effort prevents the full psychical elaboration of the stimulus, and consequent production of emotion, and therefore the galvanic reaction is diminished.

The effects of different stimuli vary in different individuals. In some there is a greater reaction to actual sensory stimuli, such as pin-pricks and burns, than to stimuli involving the ideational processes, such as the expectation of a burn or the recalling of unpleasant memories. These differences appear to have some diagnostic value. It is difficult at present to compare quantitatively the reactions of different individuals, as the electrophysiological conditions are rarely comparable, but general conclusions can be drawn from the work of different observers. In respect to data concerning the latent period the conditions are more comparable. They can be recorded with accuracy in the case of sensory stimuli, but in the case of stimuli where associative elaboration takes place it is impossible to say exactly when the emotive effect begins. All observers are agreed that the latent period in healthy persons may vary from one to five seconds. Peterson and Jung [28] have recorded that the latent period is lengthened in cases of dementia præcox, with katatonia. Abbott and Wells [1], in an investigation on maniacal-depressive stupor, have reported that "the latent times to sensory stimuli observed under stupor are within normal limits," and that the "perceptive processes in conditions of maniacal-depressive stupor are but slightly lengthened from the normal. The retardation may lie in a lessened complexity of the associations formed, or in the slower and feebler conversion of these associations into their motor expressions.

An important question is as to whether there is any correspondence between intellectual development and the quality and quantity of the psychogalvanic reflex. There certainly appears to be some correlation between intellectual development and the reactions to the two different classes of stimuli. The greater the intellectual development the more pronounced are the reactions to ideational stimuli. The same condition exists in cases of anxiety neurosis; whereas in cases of conversion hysteria the reactions to sensory stimuli appear to prevail over those due to ideational stimuli, but there is not sufficient data forthcoming to decide this with any certainty. M. D. Waller [52], as a result of her investigations on seventy-three medical students, concludes that intellectual efficiency as judged by examination results is associated with higher nervous sensitiveness in the psycho-galvanic reflex. It is certainly my experience that the reactions in subjects of poor intellect, as evidenced by their low standard at school, are rarely so marked as in my intellectual subjects, and I have attributed this to the fact that the intellectual subject has usually a much greater control over his emotions and has a wider range in the association of ideas. Claparède [5] has reported experiments on four idiots of the lowest grade, in whom no psycho-galvanic reflex of any kind was obtained even after painful stimuli, in spite of the fact that the stimuli were perceived and were responded to by marked muscular reactions.

Gregor and Gorn (14) disagree with Claparède, and quote one case in whom they obtained normal reactions to sensory stimuli.

The chief work on the psychoses has been done by Peterson and Jung [28], Ricksher and Jung [34], and Gregor and Gorn [14]. Peterson and Jung have reported experiments on eleven cases of dementia præcox. They observed nothing especially noteworthy in the hebephrenic type, but in the katatonic forms, especially the acute forms, they observed variations from the normal, both in the quality and quantity of the reflex, the chief observation being that there was almost no reaction at all in acute katatonic stupor. They consider that this result is due to the fact that "the entire psychic activity is bound up with the morbid complex." This explanation might be plausible if it were not for the fact that a similar result can be obtained in other forms of insanity, arteriopathic dementia for example, and according to Claparède in idiots, so that this result would seem to be more in accord with the auto-intoxication theory and deterioration of the cerebral cortex. Gregor and Gorn have reported similar results in dementia præcox. They found that the reactions became less as the disease advanced, but that the reactions reappeared as the patients began to come out of the stupor, and became greater as the patients began to improve. On the other hand, in transitory psychogenic stupors the reactions were normal.

The reactions were also small in cases of hebephrenia, and the reactions to pain stimuli were greater than those to other stimuli, whilst in hebephrenic maniacal excitement the reactions were practically absent. A similar picture was obtained in cases of general paralysis. In manic-depressive cases the reaction was reduced at the height of the depressive phase, but in all cases of mania there was a very marked reaction. Gregor and Gorn concluded that "the observation of the psychogalvanic reflex has a definite clinical value: (1) in judging the effects of impressions in stuporose and inaccessible individuals; (2) in distinguishing benign psychasthenic 'regression' from similar conditions in the course of progressive maladies; (3) the objective characterization of affective conditions as a criterion of affective sensibility; (4)

the prognosis of melancholia and katatonia; (5) distinguishing maniacal states due to paralytic or hebephrenic excitement; (6) the diagnosis of malingering."

In conclusion, it can only be said that a very good case has been made out for further investigation into the psychological study of the psychogalvanic reflex. The observations so far recorded are small and the opinions of the observers are often conflicting. But it would seem that we have in the psychogalvanic reflex a valuable objective sign, which may be able to help us in the elucidation of the many unsolved problems in psychological medicine, and which we cannot afford to disregard.

REFERENCES.

[1] Abbott, E. S. and Wells, F. L. "Psychogalvanism in the Observation of Stuporose Conditions," *Psychol. Rev.*, 1919, vol. xxvi, p. 5.

[2] Aebly, J. "Zur Analyse der physikal. Vorbedingungen des psychogalv. Reflexes mit exosomatischer Stromquelle," *Inaug. Diss.*, Zürich, 1910.

[3] Albrecht, O. "Exper. Unters. über die Grundlagen der sogenannten galv. Hautelektrizität," *Monatschr. f. Psychiat. u. Neurol.*, 1910, Bd. xxvii, S. 365, 439, 552.

[4] Binswanger, L. "Ueber das Verhalten des psychogalv. Phänomens beim Assoziationsexperiment," *Journ. f. Psychol. u. Neurol.*, 1908, Bd. x, S. 149; Bd. xi, S. 65, 133.

[5] Claparède, E. "Sur le phénomène psycho-électrique," *Extrait des Arch. des Sc. physiques nat.*, 1911, t. xxxi, p. 379.

[6] Du Bois Reymond, E. "Untersuchungen, &c.," 1849, Bd. ii, S. 261, 276.

[7] Féré, Ch. "Note sur les modifications de la résistance électrique sous l'influence des excitations sensorielles et des émotions," *Compt. rend. de la Soc. de Biologie*, 1888, p. 217.

[8] Fürstenau, R. "Die Stellung der menschlichen Haut in der elektrischen Spannungsreihe," *Zentralb. f. Physiol.*, 1906, Bd. xx, S. 194.

[9] Gildemeister, M. "Ueber die physikalisch-chemischen und physiol. Vorgänge im menschlichen Körper, auf denen der psychogalv. Reflex beruht," *Münch. med. Wochenschr.*, 1913, Bd. xliii, S. 2389.

[10] *Idem.* "Ueber die im tierischen Körper bei elektrischer Durchströmung entstehenden Gegenkräfte," *Pfluger's Arch.*, 1913, Bd. cxlix, S. 389-401.

[11] *Idem.* "Der sogenannte psychogalvanische Reflex und seine physikalisch-chemische Deutung," *Pflüger's Arch.*, 1915, Bd. clxii, S. 489-506.

[12] *Idem.* "Über elektrischem Widerstand, Kapazität und Polarisation der Haut," *Pflüger's Arch.*, 1919, Bd. clxxvi, S. 84-105.

[13] Girard, P. "Sur les relations osmotiques des globules rouges avec leur milieu, &c.," *Compt. rend. de l'Acad. des sciences*, 1913, t. clvi, p. 1401.

[14] Gregor, A. and Gorn, W. "Zur psychopathologischen und klinischen Bedeutung des psychogalv. Phänomens," *Zeit. f. d. ges. Neurol. u. Psychiat.*, 1913, Bd. xvi, S. 1-104.

[15] Gregor, A. and Loewe, S. "Zur Kenntnis der physikal. Bedingungen des psychogalv. Reflexphänomens," *Zeit. f. d. ges. Neurol. u. Psychiat.*, 1912, Bd. xii, S. 411-446.

[16] Hallion, L. and Comte, Ch. "Sur les réflexes vasomoteurs bulbomédullaires dans quelques maladies nerveuses," *Arch. de Physiol.*, 1895, t. vii, p. 90.

[17] Hermann, L. *Handbuch der Physiologie*, 1879, Bd. i, S. 224.

[18] Hermann, L., and Luchsinger, B. "Ueber die Secretionsströme der Haut bei der Katze," *Pflüger's Arch.*, 1878, Bd. xvii, S. 310.

[19] Knauer, A. "Kritische Bemerkungen zu O. Veraguth's Buch, 'Das psychogalvanische Reflexphänomen,'" *Journ. f. Psych. u. Neur.*, 1909, Bd. xxiii, S. 1-2.

[20] Leduc, S. "Les ions et les médications ioniques," 1906, Paris.

[21] Leva, J. "Ueber einige körperliche Begleiterscheinungen psychischer Vorgange, mit besonderer Berucksichtigung des psychogalv. Reflexphänomens," *Münch. med-Wochenschr.*, 1913, Bd. xliii, S. 2386.

[22] Markbreiter, R. "The Effect of Atropine on the Emotive Response" (in collaboration with A. D. Waller), *Proc. Roy. Soc.*, 1919, B, vol. xci, p. 40.

[23] Moravcsik, M. E. "Experimente über das psychogalv. Reflexphänomen," *Journ. f. Phys. u. Neur.*, 1912, Bd. xviii, S. 186-199.

[24] Muller, E. K., cited by Peterson [27] from *Compt. rend. de la Soc. helvétique des sciences nat.*, September, 1904.

[25] Muller, H. "Exp. Beitrage zur physikal. Erklarung der Entstehung des psychogalv. Phänomens," *Inaug. Diss.*, Zürich, 1909.

[26] Muller, V. J. "Zur Kenntnis der Leitungsbahnen des psychogalv. Reflexphänomens," *Monatschr. f. Psychiatrie*, 1913, Bd. xxxiii, S. 235.

[27] Peterson, F. *Brit. Med. Journ.*, 1907, vol. ii, p. 804.

[28] Peterson, F., and Jung, C. G. "Psycho-physical Investigations with the Galvanometer and Pneumograph," *Brain*, 1907, vol. xxx, pp. 153-218.

[29] Peterson, F., and Scripture, E. W. "Psycho-physical Investigations with the Galvanometer," *Journ. of Nervous and Mental Diseases*, 1909, vol. v, p. 426.

[30] Philippson, M., and Menzerath, P. "Analyse physiologique du phénomène psycho-électrique," *Arch. Intern. de Physiol.*, 1914, t. xv, p. 1-30.

[31] Piéron, H. "Les variations physiogalvaniques comme phénomène d'expression des émotions," *Revue de Psychiatrie*, 1910, p. 486-506.

[32] Prince, M., and Peterson, F. "Experimentelle Unters. über psychogalv. Reaktionen von mitbewussten (unterbewussten) Vorstellungen in einem Falle vielfacher Personlichkeit," *Journ. f. Psychol. u. Neurol.*, 1908, Bd xiii, S. 249-262.

[33] Radecki, W. "Recherches expérimentales sur les phénomènes psychoélectriques," *Arch. de Psychologie*, 1911, t. xi, p. 209-295.

[34] Ricksher and Jung, C. G. "Investigations on the Galvanic Phenomenon," *Journ. of Abnormal Psychol.*, 1908, vol. ii, p. 5.

[35] Schwartz, cited by Philippson and Menzerath [30], from *Zentralb. Physiologie*, 1913, Bd. xxvii, S. 734.

[36] Sidis, B. "The Nature and Cause of the Galvanic Phenomenon," *Journ. of Abnormal Psychol.*, 1910, vol. v, p. 74.

[37] Sidis, B. and Kalmus, H. T. "A Study of Galvanometric Deflections due to Psycho-physiological Processes," *Psychol. Review*, 1908, vol. xv, p. 391, and 1909, vol. xvi, p. 1-35.

[38] Sidis, B., and Nelson, L. "The Nature and Causation of the Galvanic Phenomenon," *Psychol. Review*, 1910, vol. xvii, pp. 98-146.

[39] Sommer, R. "Zur Messung der motorischen Begleiterscheinungen psychischer Zustände," *Beiträge zur psychiatrischen Klinik*, 1902, Bd. i, S. 143.

[40] Starch, D. "Mental Processes and Concomitant Galvanometric Changes," *Psychol. Review*, 1910, vol. xvii, p. 19.

[41] Sticker, G. "Ueber Versuche einer objectiven Darstellung von Sensibilitätsstorungen," *Wiener klinische Rundschau*, 1897, Bd. xxx, S. 497.

[42] Tarchanoff, J. "Ueber die galvan. Erscheinungen," etc., *Pflüger's Arch.*, 1890, Bd. xlvi, S. 46.

[43] Veraguth, O. "Das psychogalvanische Reflexphänomen," Berlin, 1909, Verlag von S. Karger.

[44] Vigouroux, R. "Sur le rôle de la résistance électrique des tissus dans l'électro-diagnostic," *Compt. rend. de la Soc. de Biologie*, 1879, t. xxxi, p. 336.

[45] Waller, A. D. "Sur la mesure de l'émotivité nerveuse," *Extrait du Bull. de la Soc. Roy. des Sci. méd. et nat. de Bruxelles*, 1919, t. vi, p. 2.

[46] WALLER, A. D. "Calorimetry by Surface Thermometric and Hygrometric Data," *Journ. of Physiol*, 1894, vol. xv, p. 25.

[47] *Idem.* "Demonstration of the Psychogalvanic Phenomenon," *Journ. of Physiol.*, 1918, vol. vi.

[48] *Idem.* "The Galvanometric Measurement of 'Emotive' Physiological Changes," *Proc. of the Roy. Soc.*, 1918, B, vol. 90, p. 215.

[49] *Idem.* "Periodic Variations of Conductance of the Palm of the Human Hand," *Proc. of the Roy. Soc.*, 1919, B, vol. 91, pp. 17-31.

[50] *Idem.* "The Influence of Drugs upon the Electrical Conductivity of the Palm of the Hand," *Proc. of the Roy. Soc.*, 1919, B, vol. 91, pp. 32-40.

[51] *Idem.* "Mensuration de l'émotion nerveuse chez l'homme," *Extrait du Bull. de l'Acad. Roy. de Méd. de Belg.* 1919.

[52] WALLER, M. D. "Emotive Response of Seventy-three Medical Students," *Lancet*, 1918, vol. i, p. 510.

[53] WEISS, cited in Guilleminot's "Electricity in Medicine," p. 202.

Section of Neurology of the Royal Society of Medicine.

President—Dr. W. ALDREN TURNER, C.B.

Forms of Peripheral Neuritis among Troops serving with the Egyptian Expeditionary Force, 1915-19.[1]

By F. M. R. WALSHE, M.D.

DURING the recent campaign in Egypt and Palestine undoubtedly the commonest organic nervous disease among the troops was multiple neuritis. It was also the only one that could definitely be said to be associated with the prevailing conditions of active service. Post-diphtheritic paralysis formed the bulk of the cases, and it is to these that my last remark applies.

During the whole period of my service from 1915 to 1919, I saw no case of multiple neuritis unquestionably due either to dysentery or to malaria, nor did any instance of acute febrile polyneuritis as it has been described by Gordon Holmes or Rose Bradford come under my observation. Finally, there was no beri-beri in Egypt or Palestine, apart from imported cases from Mesopotamia and a few in 1915 from Gallipoli.

I propose to confine my remarks this evening to a certain group of cases of multiple neuritis, and, since many of the symptoms of this malady are common to all its ætiological varieties, it will save you much wearisome and familiar detail if I single out for remark such noteworthy features of my cases as may be more or less new to you, and that may throw some light upon the origin of post-diphtheritic paralysis.

Of this, I saw over 160 cases. They fell into two groups: the first consisting of the ordinary type following faucial diphtheria; the second, and in some ways the more interesting group, associated with extra-faucial infections of superficial skin lesions and of wounds by the diphtheria bacillus. The opportunity of seeing so large a series was unique, for over sixty came under observation.

---

[1] At a meeting of the Section, held January 8, 1920.

However, before referring to the characteristic clinical picture presented by these I should like to make some general observations on the order of appearance of the signs and symptoms of multiple neuritis. Having access to a large Isolation Hospital, I was able to observe continuously a number of cases of diphtheria until their discharge to Convalescent and Command Depots. Many were examined almost daily from the date of admission, and others from the appearance of palatal paralysis in the second and third weeks of their illness. Multiple neuritis developed in several of these while under observation, and contrary to expectation, I found that diminution and abolition of the knee- and ankle-jerks were by no means the initial signs of the malady. Subjectively, aching pains in the legs on exertion, painful cramps in the calves during the night, numbness of the feet and, objectively, tenderness of the calf and plantar muscles on pressure, with a remarkable facility of the tendon-jerks, were the earliest phenomena. In six cases I was satisfied that the knee- and ankle-jerks underwent a definite preliminary exaltation before they began to grow sluggish. In many cases the ankle-jerks became feeble while the knee-jerks were still brisk, and they always disappeared earlier than the knee-jerk.

The point is, perhaps, of minor clinical interest. Nevertheless, in the circumstances in which the observations were made there was a definite value attaching to them. For in dealing with convalescents from diphtheria, the eliciting of the knee-jerks was commonly regarded as giving a clean bill of health to the patient in this respect, and as being a sufficient reason for disregarding any subjective symptoms of which he might complain. But in my experience this test was by no means adequate to exclude an early polyneuritis, and thus many men were sent prematurely to duty only to be readmitted to hospital within a week or two with a fully developed polyneuritis.

To return to the question of post-diphtheritic paralysis: during the winter 1916-17 and onwards there was an epidemic of diphtheria in the field and throughout the lines of communication, which extended from Palestine to the Delta. Although of mild character, on the whole it was fairly heavy in incidence, and, associated with it, I saw numerous cases of the ordinary type of post-diphtheritic paralysis. I am not able to give even an approximate estimate of the total case incidence in this outbreak, nor of the proportion of cases of diphtheria in which nervous complications ensued, but I think that the latter was high, and I ascribe this to two causes: first, the small dosage of antitoxin, a single dose of from 4,000 to 6,000 units being the rule; and secondly, the premature return of the patients to conditions of physical activity. Often within a month of an apparently mild attack of diphtheria, a man resumed route marching at a Command Depot, only to fall out with pains and weakness of the legs on his first march. In such a patient, one could watch the development of a full-blown polyneuritis, which disabled him for at least six months. In many of these men the knee-jerks were still obtainable on readmission for a short period. Further, many mild cases were, I am sure, never recognized as such but passed under various diagnoses—for example, tabes dorsalis, neurasthenia, and "D.A.H.," and thus any estimate of the total number was impossible.

Of the cases of post-diphtheritic paralysis following the ordinary faucial inceptions, there is little of particular interest to record, except possibly the large proportion in which the usual palatal and accommodation pareses were followed by a fully developed polyneuritis, probably for the reasons I have already given.

Early in 1917 cases of a mild type of multiple neuritis began to appear. They were not associated with any history of sore throat, nor with any other evidence of faucial diphtheria. The common ætiological factors seemed absent also, and the only feature common to the cases was the recent occurrence of a crop of "septic sores," except in one case, in which there had been a chronic suppuration in a flesh wound of the arm of some months' duration. The significance of this history was not recognized at once. And although the possibility of a secondary diphtheritic infection of the skin lesions was thought of, it was dismissed because the multiple neuritis was neither preceded nor accompanied by palatal or accommodation paralysis. In fact, the first ten or twelve cases that I saw had no cranial nerve symptoms whatever. In every instance their general condition was good, though there was in all of them a mild degree of tachycardia ranging from 80 to 100 when at rest.

However, some light was thrown on the question in the April of that year by a case which I have already recorded elsewhere, which made it appear highly probable that diphtheria lay at the bottom of all these obscure cases of multiple neuritis. The case was this: a medical officer attached to the same unit as myself performed a tracheotomy upon a fatal case of laryngeal diphtheria. He was not aware of having scratched himself at the time, but next morning he noticed a painful bleb upon the dorsum of the first phalanx of the right thumb. This broke down leaving a sore raw surface. The ulcer rapidly spread over the surface of the thumb and ultimately reached the size of 1 in. by ½ in. It was covered by a greyish slough and exuded a scanty watery secretion. On the fourth day he began to feel ill and to shiver. There was a lymphangitis up the arm and the axillary glands were swollen and tender. On the sixth day the diphtheria bacillus was isolated in pure culture from the wound and he was admitted to hospital. During the second week of the illness, severe symptoms of toxæmia appeared: vomiting, cardiac irregularity and albuminuria, and persisted for about ten days. Improvement then set in, the wound began to heal, and in the seventh week he was discharged to convalescence. The knee-jerks were still brisk at this time. During the following two weeks he improved considerably, was able to walk uphill and to swim several hundred yards in the sea without undue fatigue, but from the seventh week onwards he had begun to notice that the thumb over and around the healed scar was very numb to touch and even to pressure. This impairment of sensation spread over the thumb and radial half of the hand, then invaded the rest of the hand and the forearm. Simultaneously his hand became very clumsy in fine movements, there was a constant feeling of pins and needles in the fingers and his handwriting became very untidy and difficult to read. His grasp was weak. About a week after his return to duty, that is, in the tenth week, he began to find that walking upstairs was very exhausting and required effort; his knees felt as though they might give way under him. Writing became quite impossible not only on account of the ataxy but because of painful cramps in the small hand and forearm muscles. There was no cranial nerve involvement. Twelve weeks after the original infection he was readmitted to hospital. On examination I found a well marked polyneuritis, with absent tendon-jerks, moderate weakness, tenderness of muscles and the characteristic sensory loss. But in addition to this, the condition of the right arm at once attracted attention. It was considerably weaker than its fellow. Its muscles were more flabby and the distal half of the limb was distinctly thinner. All fine movements of the hand and the movements of the proximal segments of the limb were ataxic. There was complete loss to touch over the hand and the distal third of the forearm and gross loss of other forms of cutaneous sensibility.

Here, then, was a case undoubtedly diphtheritic in origin, in which with the absence of palatal and accommodation paralysis, there were an initial local onset of nervous symptoms related to the site of the infective focus, and a multiple neuritis.

A short time after, a second and similar case was observed, and this is the last case I shall quote: The patient had received a flesh wound in the left lumbar region at the battle before Gaza in April, 1917. Although freely laid open early in the course of treatment and subsequently treated with every new and old-fashioned antiseptic, it had resisted healing. During August he was allowed up more freely, but as soon as he began to walk about he complained of an increasing weakness, of "pins and needles," and of cramp in his legs. A provisional diagnosis of syphilitic ulceration and early tabes dorsalis was made and the man was transferred to my care. From a small unhealed patch at the centre of the keloid scar, the diphtheria bacillus was isolated in pure culture, while clinical examination revealed a fully developed multiple neuritis. In this case there was neither local nor cranial nerve affection. I say there was no local palsy, but there was a large area of sensory loss surrounding the wound, which could not be accounted for by the local surgical condition.

During the summer of 1917, several pathologists were reporting the presence of "diphtheroid rods" in septic sores occurring in areas where faucial diphtheria was present, and at the same time numerous cases of polyneuritis following crops of septic sores came under my observation. I may say that by septic sore I refer to a condition in which small areas of superficial ulceration appeared upon the dorsum of the hand, the extensor surface of the forearm and on exposed parts of the leg round the knee. This condition was extremely prevalent throughout the campaign, especially in mounted troops when on trek in the desert. They began to concern me because from the time that diphtheria became prevalent these skin lesions were undoubtedly associated with a considerable amount of multiple neuritis among the troops. Unfortunately, in the majority of cases the primary lesions were all healed by the time the cases came to me with nervous symptoms, but in addition to the two cases already described I ultimately saw nine cases in which the cautious pathologist was able to isolate "a diphtheroid organism morphologically indistinguishable from the Klebs-Loeffler bacillus."

In all, I examined over sixty such cases of polyneuritis, and in addition to their close association as to time and place with an outbreak of diphtheria and of diphtheritic paralysis, and to the finding of the organism in several of them, certain clinical signs that they showed convinced me that they were true post-diphtheritic paralyses following cutaneous infections. Some new features were seen later in the series that the first cases had not shown, and an analysis of the whole number, made when I had observed thirty and confirmed by a second thirty seen later, revealed that the complete symptom complex consisted of three elements:—

(*a*) An initial local paresis related anatomically to the site of the infective focus.

(*b*) A paralysis of accommodation, and

(*c*) Polyneuritis.

The local symptoms usually preceded by two or three weeks the visual and general symptoms. They occurred in about a third only of the total number. The accommodation paralysis was also present in but a third of the cases, while the polyneuritis was present in all. In the majority of instances multiple septic sores on the limbs preceded the onset of nervous symptoms, but in several the infective focus was single—a gun-shot wound or a boil—and it was in these that the local paralysis was most commonly seen. In the first thirty cases, eight had a single primary focus, and in six of these there was an initial local onset. In none of the number was palatal paralysis observed, and in none were diphtheria bacilli found in the throat.

A brief consideration will show that these three elements are also found in post-diphtheritic paralysis as it follows the ordinary faucial infections, for the palatal paralysis is a true local palsy; the musculature of the soft palate is clearly closely related both in the matter of proximity and in the more important respect of innervation to the local lesion on the tonsils and fauces. This formula of three elements in the clinical picture of a fully developed diphtheritic paralysis may, therefore, be applied to any case, no matter where the infective focus may be. The question at once arises as to the origin of the nervous complications of diphtheria, and the path by which the toxin reaches the nervous system. The view commonly held is that diphtheritic paralysis is the result of a blood-borne toxæmia of the nervous system in which certain centres in the brain-stem are picked out by the selective action of the toxin. And, since experimentally inoculated animals do not show cranial nerve involvement as a part of their nervous symptom-complex, the further opinion has been expressed that in animals, on the other hand, the spinal nerve centres are specially attacked.

It seems, then, reasonable to say that the cases I have briefly described do not accord with this view, and, indeed, cannot be explained by it. For if the nervous lesion in diphtheria be the result of a selective blood-borne toxæmia, its clinical manifestations should not vary according to the site of the infective focus, but should be constant and unvarying, except in intensity. It appears to me that the following view best accounts for the presence of the three clinical elements I have described; it is of course an hypothesis that as yet lacks both experimental and pathological confirmation; it is as follows: That the initial local palsy is the result of the passage of the toxin from the infective focus to the central nervous system, brain-stem or spinal cord, along the perineural lymph channels of the peripheral nerves innervating the site of the local lesion. This is the process so extensively investigated by Orr and Rows and named by them "lymphogenous toxi-infection of the nervous system." The suggestion is therefore merely an extended application of a known pathological process. This is not to say that the symptoms are caused by an ascending neuritis, for we know from Orr and Rows that the nerve, as far as its nervous elements are concerned, is commonly intact, and that the lesion is central and is situated in that segment of the central nervous system from which the nerve arises. We may thus explain equally well the initial palatal paralysis of a case of faucial diphtheria, or the initial sphincter disturbances of a case of diphtheritic infection of an ulcer in the perineum.

Accommodation paralysis and polyneuritis are common to all types. For the former, therefore, we may still invoke the hypothesis of selective action, while the latter may be regarded as the expression of the general toxæmia as it affects the nervous system, while in both instances the toxin probably reaches the nervous system by the blood-stream.

It is of interest to determine whether the initial local symptoms indicate a peripheral or a central lesion of the nervous system, and it does not appear to me that the palatal symptoms of the ordinary type throw much light on this point. In the extra-faucial cases it is equally difficult to decide because the presence of a multiple neuritis masks any distinctive characters the initial palsy may possess. However, I was so fortunate as to see one case in which this was in a region usually unaffected in multiple neuritis. The case was one of ulceration in the perineum, arising as a septic sore (and later, presumably, secondarily infected by the diphtheria bacillus), which was followed by a local, an accommodation and a generalized nervous symptomatology. The local symptoms were sensory loss over the territory of the lower four sacral posterior roots on both sides and definite loss of control over the bladder, which, I think, can only be explained by supposing that the lesion was central and not merely an ascending neuritic process in the afferent nerves of the region.

I also saw a case—and one has been recorded by Oppenheim—of a streptococcal infection of the tonsils and fauces in which palatal paralysis and a multiple neuritis supervened. In both repeated search failed to demonstrate the presence of the diphtheria bacillus. Oppenheim remarks of his case that this clinical picture cannot be regarded as absolute proof of a diphtheritic infection, and I would suggest that these cases tend to confirm the view of the origin of the initial local phenomena that I have put forward. Several recent case reports in German periodicals of extrafaucial diphtheria and a resulting nervous involvement also bear me out in this respect.

In considering post-diphtheritic paralysis from the point of view I have put forward this evening, one cannot escape the analogy it presents to tetanus. Meyer and Ransom have divided the symptoms of this into three groups.

(*a*) *Local tetanus*, in which, after prophylactic inoculations of tetanus antitoxin, the first symptoms appear in the immediate vicinity of the infective focus.

(*b*) *Specific tetanus*, in which, irrespective of the site of the infected focus, there is spasm of certain muscles—namely, trismus; and

(*c*) *Generalized tetanus*.—If the amount of toxin be considerable a generalized tetanus is superadded to the two preceding forms.

After intravenous injections of tetanus toxin into animals, local tetanus is not seen; similarly, after intravenous injection of diphtheria toxin into experimental animals, the paralysis is generalized and presents no local phenomena. We may, therefore, compare the local, specific and generalized forms of tetanus with the palatal, ocular and generalized nervous symptoms of faucial diphtheria—the palatal palsy being the local element, the accommodation affection the specific element, while the polyneuritis corresponds to generalized tetanus.

In conclusion, I must ask your indulgence for the somewhat broad manner in which I have dealt with my subject. There are many gaps, I know, on the theoretical side of my paper. I have merely tried to describe a somewhat unusual type of peripheral neuritis, and to indicate briefly the theoretical considerations to which its study gave rise, and its bearing on the pathogenesis of post-diphtheritic paralysis.

I know also that this is by no means a comprehensive review of the various forms of peripheral nerve disease that one saw in Egypt. However, the rest of these were merely fortuitous in their occurrence and were in no way peculiar to that field of operations. I have thought it best, therefore, not to attempt completeness, but only to deal with what seemed characteristic of the campaign in which I served.

DISCUSSION.

Dr. PHILIP MANSON-BAHR: I feel I am here under false pretences; what I am going to say as a sort of confirmation of Dr. Walshe's paper is the result, not of my own work, but that of my friend and late colleague, Dr. Craig, of Kendal. It was during the advance through the Sinai desert, and especially on the line of the Wady Guzze before Gaza, that "desert sores," or "septic sores," as they were called, first began to claim serious attention. Now from what we know this desert sore is probably synonymous with the "veldt sore" of South Africa and the "Barcoo rot" of Australia; at any rate it has something to do with desert conditions, and is quite distinct from the Oriental sore due to *Leishmania tropica.* Now it was noted by Craig that in the Egyptian Expeditionary Force the majority of units afflicted with these sores were mounted troops, or those associated with camels, and he concluded that as the clinical appearances of the lesions were so characteristic they must have a peculiar and specific cause. The sores affected those parts of the body exposed to injury—for instance, the hands, knees, and face. In mounted troops who rode in "shorts," as they did at that period, the sores were apt to form over the points of friction at the knee. However, in many cases they supervened *de novo* after no obvious sign of any injury. The onset of the sore is sudden and very painful, quite out of proportion to the size of the lesion; within a few hours a vesicle forms filled with serous, or it may be hæmorrhagic, fluid, collecting generally round a hair follicle. The vesicle soon bursts and leaves behind a raw area generally covered with a pearly-grey membrane and moist with exuding serum. The edges of the skin soon become undermined and the ulcer spreads in a peripheral direction and soon becomes punched out, chronic, and most intractable. The surrounding skin is livid and blue. Apart from the pain and disability they caused, the amount of debility, cardiac irregularity, and often anæmia, which existed in association with the sores, were sufficient to attract general attention and speculation as to the specific cause. Early in 1917 Dr. Charles Martin had reported the isolation of a "diphtheroid" bacillus from one of these sores, a fact which he did not then regard as significant, but rather he looked upon the lesions as being either streptococcal or staphylococcal in origin. This line Craig followed up, and in a short time was able to isolate diphtheroid bacilli, indistinguishable morphologically from the Klebs-Löffler bacillus, from 129 out of 197 of the sores he examined, that is, in 67·5 per cent. Making due allowance for the fact that many of his patients had been extensively treated with all manner of antiseptics, he reckoned that the percentage of successful operations should have been much higher. Almost pure cultures of Klebs-Löffler bacilli were obtained by Craig and myself from the serum which exuded from the vesicles on Löffler's glucose serum, but the longer the ulcer had persisted, the more difficult it was to isolate the organism and the more numerous were staphylococci and streptococci. There was no doubt that they were typical Klebs-Löffler bacilli; they conformed to all the well-known tests, produced death in thirty-six hours when injected into guinea-pigs or quails, with hæmorrhagic peritonitis and lesions of the suprarenals; on the other hand, these animals could be efficiently protected by a simultaneous injection of antidiphtheritic serum. At the same time as these sores were so prevalent a large number of cases of faucial diphtheria were occurring, and most of our energies, as pathologists, were directed to cultivating the Klebs-Löffler bacillus in throat swabs by the thousand. But I must confess that we were more fortunate in obtaining a higher percentage of positive isolations from desert sores than from throat cultures. When General Allenby came out, he took, amongst other things, more than an active interest in these desert sores, as they were causing a great amount of disability; in some units as much as 40 per cent. of the men were unfit for hard labour and had their hands swathed in bandages. There was at this time, for all-sufficient reasons, a considerable amount of official reticence in acknowledging the importance of Craig's discovery. It was thought that the absence of fresh elements, and especially of beer, from the dietary had a mysterious influence upon these sores, so General Allenby caused all the beer in Egypt to be sent up the line. This had the effect of raising the spirits of the men, but it did not heal their sores. At this time we began to collect together a considerable amount of evidence of the association of these sores with paresis or neuritis of various parts of the body, as Dr. Walshe has just described. For instance, I remember the case of a corporal of the H.A.C. who was covered with these sores, who had neuritis of both legs, with lack of knee-jerks, hyperæsthesia of his calves, and great ataxia. He came down the line with a provisional diagnosis of tabes. Another I remember with paralysis of the palate following sores on the eyebrows and cheeks, and paresis and numbness of the arms, with multiple sores on the fingers and hands. Probably paralysis was much more common than was supposed, and in many cases it had been overlooked or attributed to presumably untreated faucial diphtheria. The effect of injection of 4,000 units of antidiphtheritic serum in the neighbourhood of these sores was almost magical; raw patches and ulcers which had persisted for months, nay, even years, after treatment with every form of antiseptic lotion and paste, healed up perfectly with sound firm skin within a week or ten days. I myself am absolutely convinced that this was so, though no success was obtained in getting this mode of treatment recognized officially or universally adopted. The scars which remained after healing are sufficiently characteristic, and the stigmata of " desert sores " will be evident for years; there is probably not a single man who served in the Australian Light Horse or Imperial Camel Corps who does not bear one or more. The hypothesis that these sores were a primary skin infection with the diphtheria bacillus led Craig to consider that they were in some way connected with desert transport, for they ceased to be a scourge directly we had moved forward into the more fertile and cultivated portions of Palestine. Suspicion fell on horse manure as a source of infection.

At that time the desert roads were laid upon a foundation of horse dung, which in this manner afforded a harder surface than sand for travel. Finally Craig succeeded in isolating a diphtheroid bacillus from this substance, but lack of opportunity prevented him from carrying his researches any further. From the experience I have had of this subject I should not be at all surprised to learn that a considerable number of cases of tropical polyneuritis, formerly diagnosed as beri-beri, are explicable on a similar basis. Finally, I would add that the inability of some pathologists to isolate the diphtheria bacillus from these ulcers in base hospitals in no way militates against Craig's work. By the time they reached the base these sores had been contaminated and invaded by all sorts of organisms, such as streptococci, which therefore cannot be considered the actual ætiological agent or the cause of the debility and neuritis associated with them. As an illustration I show a photograph of a Mesopotamian case recently under my care in which a sore suspected of being cutaneous leishmaniasis had persisted for two and a half years, and which instantly healed up after injection of antidiphtheritic serum.

Dr. FEILING: During my three years' experience in Mesopotamia I saw many cases of polyneuritis of various kinds in both British and Indian troops. On the whole it was not a very common disease there, at least amongst the British. I wish first to say a few words on the subject of beri-beri. While having the greatest admiration for the work done by many observers in attacking the problem of its ætiology from the viewpoint of a dietetic deficiency, I feel obliged to demur to this being the only cause or in all cases any essential factor at all. Beri-beri after all is at present only a clinical term and different infections or toxins can produce in the field of polyneuritis clinical pictures which are very difficult, if not impossible, to differentiate on clinical grounds alone. I would like to illustrate this by brief reference to two epidemics of polyneuritis with which I became acquainted in Mesopotamia. One affected a number of Chinese labourers. These men were all of a fairly superior class, skilled carpenters mostly, and were housed in two large huts. The cases were confined to men in *one* of these huts only. Their rations had been expressly designed to contain as many so-called "anti-beri-beri vitamines" as possible. They received good pay and were able to, and did, supplement their rations by extensive purchases in the local bazaars of such fresh food as fish, eggs, chickens and vegetables; I myself often saw them returning from market loaded with such purchases. Clinically the cases were typical beri-beri; many of them of the so-called "wet" or œdematous type. Another example of an epidemic of polyneuritis, which illustrated the difficulties of diagnosis and its consequent evils, was that in which a large number of cases of a severe form of polyneuritis occurred in a detachment of British troops occupying an isolated post in the desert. These cases were diagnosed as beri-beri, and many of them ultimately found their way to Bombay. There fuller investigations were undertaken with the result that arsenic was found in large quantities in the hairs of many of these cases.

Inquiries were instituted and it was eventually discovered that a barrel of white arsenic had by some means been included amongst the barrels of bleaching powder used for water disinfection, and that these unfortunate men had been steadily disinfecting their water supply for some time with white arsenic. With regard to *dysenteric* neuritis, I have seen a number of such cases in which a dysenteric infection, generally of the bacillary type, was followed by polyneuritis. Less frequently it occurred amongst amœbic cases. It has been suggested, though on no very clear grounds, that the emetine given therapeutically was the cause of the neuritis in the amœbic cases. I saw one rather remarkable example of this condition. The patient was a young officer, who was admitted to hospital for fever and shortly afterwards operated on and a large liver abscess evacuated; the *Entamœba histolytica* was found in the pus from the abscess. During convalescence he developed a very severe polyneuritis which in its clinical features was quite indistinguishable from beri-beri. *Typhus* fever has been the cause of a certain small number of cases. In one of such the signs suggested that the infection had fallen on the ganglion cells of the cervical enlargement of the cord rather than on the more distal part of the motor neurone, for the principal feature of the case was extreme atrophy of the muscles of the shoulder girdle, the whole picture closely resembling that produced by an acute anterior poliomyelitis of the upper extremities. The *influenza* epidemic of 1918 was responsible for many cases of polyneuritis, many of them of a very severe type. Some of these again bore a very close resemblance to the classical picture of beri-beri. I saw one case which showed the somewhat remarkable combination of extreme atrophy of the leg, with great œdema affecting the subcutaneous tissues of the trunk only; this case also showed diaphragmatic paralysis and a severe cardiac affection; in spite of the fact that malaria became a still further complication the patient eventually recovered completely. In conclusion, I would point out that in only too many of the cases the ætiology has been a matter of grave doubt or even pure speculation.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[JULY, 1920.]

CALCUTTA UNIVERSITY
Sec. No. ....
Book No ....
LIBRARY.

BRAIN.

PART 2, VOL. 43.

APHASIA AND KINDRED DISORDERS OF SPEECH.[1]

BY HENRY HEAD, M.D., F.R.S.

PAGE
CHAPTER I.—MATERIAL AND METHODS 89
§ 1.—Serial Tests 91
(*a*) Naming and Recognition of Common Objects 93
(*b*) Naming and Recognition of Colours 96
(*c*) The Man, the Cat and the Dog Test 98
(*d*) The Clock Tests 98
(*e*) The Coin-bowl Test 101
(*f*) The Hand, Eye and Ear Tests 101
§ 2.—Further Tests employed in this Research 105
CHAPTER II.—PREVIOUS EXPLANATIONS OF APHASIA 107
§ 1.—Disorders in the use of Language, due to an Unilateral Lesion of the Brain, cannot be classed under the Categories of Speaking, Reading and Writing 108
§ 2.—These Disorders of Language are not due to destruction of Images 111
§ 3.—The "Motor" Aspect of these Disorders of Language is not a pure "Anarthria" 113
CHAPTER III.—THE NATURE OF THE FUNCTIONS DISTURBED IN APHASIA AND KINDRED DISORDERS OF SPEECH 115
CHAPTER IV.—DISSOCIATED FORMS OF SYMBOLIC THINKING AND EXPRESSION .. 119
§ 1.—Verbal Defects 121
§ 2.—Nominal Defects 127
§ 3.—Syntactical Defects 136
§ 4.—Semantic Defects 142
§ 5.—Differences between these various Forms of Disordered Speech contrasted 148
CHAPTER V.—SYMBOLIC THINKING AND EXPRESSION 157
SUMMARY 162

IT is my pleasant duty to thank the Master and Council of St. John's College, Cambridge, for selecting me to deliver this lecture. It was founded close upon four hundred years ago by the famous grammarian and humanist physician, Thomas Linacre, and I feel that I cannot bring a more suitable tribute to his memory than by attempting to penetrate the mysteries of aphasia and kindred disorders of speech.

---

[1] The Linacre Lecture for 1920.

This paper is a preliminary communication of conclusions to which I have been led mainly from the study of soldiers wounded in the war. I make no attempt here to correlate my results with those of other workers in the field; this I hope to do later in the Hughlings Jackson lecture, when I shall deal with the history of aphasia and kindred disorders. My object in the following pages is to put forward briefly a fresh conception of the clinical phenomena of loss of speech from the physiological rather than from the anatomical point of view.

Physicians first recognized that the acts of speaking, reading, and writing could be affected by a gross cerebral lesion at a time when many still believed in the "localization" of "human faculties" and "moral qualities" in various portions of the brain. At the beginning of the nineteenth century Gall had enunciated this theory, which had found wide acceptance. By a perverted process of reasoning from analogy, the under surface of the frontal lobes had been selected as the seat of the "sense and memory of words." This doctrine was hotly disputed, and by the middle of the century had begun to fall into disrepute. When, however, in 1861, Broca found that local disease of one half of the brain produced definite loss of speech, the theory of "localization" seemed to be confirmed, the more so since he placed the cause of the disorder in the third frontal convolution. The nature of the problem was, however, still obscured by the older idea that it was the "memory for words" or the "faculty of speech" which was situated in the affected parts of the brain.

But from the beginning (1868) Hughlings Jackson protested against the idea that there was a "faculty" of speech that could be destroyed by a cerebral lesion. These defects must be considered, he maintained, "on the psychical side as defects of mind, and on the physical side as defects of the nervous system." We have to consider speech on this wider basis, in order that we may be better able to see how speech is part of mind, and to get rid of the feeling that there is an abrupt and constant separation into mind and speech. "It is," he said in 1868, "the power of intellectual expression by 'movements' of any kind, which is impaired, those most special, as of speech, suffering most; those of simple sign-making, least or not at all." "The question" he adds, "is not, How is general mind damaged? but, What aspect of mind is damaged?'"

LIBRARY

Most observers continued to cling to the terms "aphasia," "alexia," and "agraphia," and many cases were published which were supposed to be examples of these conditions in isolated form. But at the same time it was recognized that the acts of speaking, reading and writing, were based on more elementary processes; these were supposed to be "images" of movement, hearing and sight.

This assumption was purely gratuitous. In 1866 Jackson pointed out that, in some cases, images were entirely unchanged; in some, on the other hand, they were grossly disturbed, and to these he applied the special term "imperception."

But, with Wernicke's description of "Sensory Aphasia," the belief that certain instances of disordered speech were due to destruction of auditory or visual images seemed to become a demonstrable fact. Broca's aphasia was attributed to disturbance of the motor mechanism, whilst the other forms were said to be due to destruction of "auditory" or "visual word centres." The terms "motor aphasia," "word deafness," and "word blindness" became firmly established, although they were no more fitted to describe the actual loss of function than "aphasia," "alexia," and "agraphia." As time went on, it was evident that the actual disorders produced by a cerebral lesion did not correspond to any of these categories. Elaborate explanations were put forward, which tended more and more to be based on mechanical diagrams, founded neither on the anatomy nor on the known functions of the brain.

Finally, Marie made a bold attempt to bring theory into consonance with clinical facts; he put forward the view that "motor" aphasia was due to anarthria or disturbance of the higher articulatory mechanism, whilst the "sensory" form was in reality an expression of lowered intellectual capacity.

I hope elsewhere to enter more fully into the history of the various views on the nature of aphasia and kindred disorders. But this short statement must suffice to introduce the observations I am anxious to put forward in this paper.

CHAPTER I.—MATERIAL AND METHODS.

An inconstant response is one of the most striking results produced by a lesion of the cerebral cortex. During our studies in sensation, we found that a stimulus, exerting a constant physical force well above the normal threshold, was sometimes appreciated and at others evoked no reply. Moreover, a graduated increase of intensity did not of necessity lead to an equivalent improvement in the answers given by the patient.

Bearing in mind this characteristic want of certainty in the reaction to measured stimuli, it seemed probable that disorders of speech due to cerebral injuries would reveal the same tendency. This is notoriously the case. It is not a sufficient test to hold up some object, and ask the patient to name it; at one time he may be able to do so, at another he fails completely. No conclusion can be drawn from one or two questions put in this way; his power of responding must be tested by a series of observations in which the same task recurs on two or more occasions.

Not only is it necessary to arrange the tests in sequence, but each set must be placed before the patient in several different ways. For example, six common objects are laid on the table in front of him, and he is asked to point to the one which corresponds to a duplicate placed in his hand out of sight. This is repeated for eighteen or twenty-four observations, so that the choice of any one object recurs three or four times in the course of the series. Then he indicates each one in turn, as it is named by the examiner, or makes his selection in answer to printed words set before him on a card. He next gives names to the various objects, one by one, and finally writes them down without saying anything aloud. The order in which the tests follow one another remains the same throughout; this alone makes it possible to draw any conclusion from the inconstant responses, which are so disconcerting, unless the answers are recorded in this manner. Moreover, this method enables us to learn how the patient responds to the same series of tests put before him in different ways.

Before we pass on to consider these tests more in detail, it is necessary to say something of the character of the patients on whom this research is mainly based. In civilian practice most of those who suffer from aphasia and kindred disorders of speech are men with arterial degeneration, and in many the blood tension is greatly increased. They are old, broken down, and their general intellectual capacity is diminished. Such patients are easily tired and are obviously unsuitable for sustained examination.

But the war brought under our care young men who were struck down in the full pride of health. Many of them were extremely intelligent, willing and anxious to be examined thoroughly. As their wounds healed they were encouraged and cheered by the obvious improvement in their condition. They were euphoric rather than depressed, and in every way contrasted profoundly with the state of the aphasic met with in civilian practice.

There is still another difference between the results produced by gunshot injuries of the head and those vascular lesions which are usually responsible for disorders of speech in the old. The missile strikes the skull from without, and even if it penetrates the brain tends to cause the greatest damage on the surface. Many vascular lesions, on the other hand, destroy the substance of the brain where the fibres are diverging or converging on their path to or from the cortical centres; a small hæmorrhage may in consequence be followed by a profound and widespread disturbance of function. But structural changes produced by a local injury to the external surface of the skull, not only cause less severe and extensive manifestations of cerebral injury, but give greater opportunity for the appearance of loss of function in dissociated forms.

Moreover, with gunshot wounds of the head, the symptoms tend to clear up to a considerable extent, however severe may have been the effect produced by the initial impact of the bullet, provided there are no secondary complications. Some aspects of the disordered function recover more rapidly than others; then it is that the various cerebral activities associated with speech, reading and writing become revealed in isolated forms. As recovery proceeds, some one of them may remain permanently defective, or in the end the patient may recover his powers to such an extent that he no longer fails to carry out the rough and simple tests which can be employed in clinical research.

§ 1.—*Serial Tests.*

Before proceeding to describe in detail the actual tests used in this research, I wish to lay stress on certain general rules necessary for their success. The patient must be examined alone, in a quiet room, apart from all distracting sights and sounds. It is of fundamental importance to record not only what he says or does but also every remark or question of the observer. As soon as it is certain that the patient understands the task he is asked to perform, each series of tests must be carried out in silence; should this rule be broken, both sides of the conversation must be recorded. It is particularly important to write down at the moment any statement which throws light on the ideas or feelings of the patient with regard to the test, or to the difficulties he experiences in carrying it out. If it consists in executing some choice to verbal command, the observer must say the words once only in the simplest and most direct manner. Should it be necessary to repeat the order at the request of the patient, the fact must be noted, so that we may learn in how far his subsequent conduct is influenced by the repetition. Between any two series of tests, it is well to permit the patient to rest or to talk freely; but as soon as a fresh set of observations has been started, all conversation should be confined to the task in hand, and every word spoken on either side must be recorded.

It is extremely important to avoid all fatigue or loss of temper. Some patients, especially the older aphasics, become depressed or angry when they fail repeatedly to carry out tests which are childishly simple. The sequence of the various sets of observations must then be rearranged, so that the next series belongs to a group that can be carried out easily. It is remarkable how quickly this restores the patient's equanimity. In all work of this kind fatigue and disappointment must be avoided by every possible means, even if necessary by terminating the sitting.

For it must be remembered that we are here dealing with a general function, and not with an affection confined to one half of the body. There are no normal parts that can be used as an indication of the patient's general condition, as was the case when we were investigating the sensory reactions of the cortex. It is not a question of local loss of attention or fatigue; the functions which are disturbed form part of the general activities of the mind.

This opens up another difficulty which was not present in our previous researches. Much in the character of the patient's answers depends on his previous aptitudes, which are entirely unknown. When testing sensation, this was of little importance, because every observation made on affected areas of the body could be compared with the response from equivalent normal parts. Since it is impossible to discover how the patient would have responded before he became aphasic, I have depended mainly on the reactions of young men wounded in the war; for, especially in the case of officers, it was possible to estimate with considerable accuracy the extent of their education, and the ability with which they had carried out the more exacting of their military duties. At the same time the profession or occupation exercised before the war frequently showed that they must have possessed faculties which were subsequently found to be grossly affected. Thus, in one case, an accountant could no longer carry out simple arithmetical operations, and a bank clerk had lost the power of adding up a column of figures with certainty.

The tests I am about to describe in detail vary greatly in the difficulty they present to persons of normal understanding. Most of them, such as naming common objects or colours, the man, cat and dog test, and the coin-bowl test, are childishly simple, and can be carried out perfectly by the stupidest individual. But many normal men are liable to make mistakes in the hand, eye, and ear test, when attempting to imitate the movements of an observer sitting opposite to him. This difficulty seems, however, to be lessened after the instruction in signalling, through which all our young officers were compelled to pass before obtaining a commission.

All the tests in any one group are not of the same order of severity. Some can be carried out with greater ease and rapidity even by normal persons, and these differences may become greatly exaggerated in pathological cases. This is particularly evident when the command requires choice between two or more courses of action. Moreover, words conveying an order always set a harder task than those which indicate an object. Told to put out his tongue, the patient may point to it, but be unable to protrude it; and yet he can do so perfectly to lick his lips.

Throughout these observations it is important to record the rapidity as well as the nature of the response. Frequently the number of erroneous answers is sufficient to show that the power to carry out some particular series of tests is defective. But occasionally every actual reply may be correct, and yet it is evident from the patient's statements and demeanour that he has difficulty in carrying out the task he has been set. A bare record of the number of times he has been successful in his choice would not reveal this defect. In many cases, therefore, it is necessary to time the interval between question and answer with a stop-watch. Speaking roughly, it will be found that the majority of rapid replies are correct, and any task performed with ease is carried out quickly.

(*a*) *Naming and recognition of common objects.*—Six objects of daily use,[1] such as a pencil, a key, a penny, a match-box, a pair of scissors and a knife, are laid on the table. Before starting the observations it is well to record the actual position in which they lie to one another from the patient's point of view. They are then screened from his sight, and a duplicate of one of them is shown to him; the screen is rapidly withdrawn, and he is asked to point to the object on the table which corresponds to the one he has just seen. From eighteen to twenty-four observations are carried out in this manner, and patient and observer remain completely silent throughout. The power to carry out this form of the test is not affected by any of the disorders of function which are the subject of this research; but it is well to begin with a task that can be performed without difficulty by the patient in order to encourage him, and at the same time to gauge his general powers of comprehension.

---

[1] Sometimes, when my patient was highly educated, I have replaced these articles of daily use by solid geometrical figures, having first ascertained that he was familiar with their proper names.

Then he is asked to point to an object on the table which corresponds to the name said aloud by the observer. This series is carried out in exactly the same order as the first and all subsequent sets of observations.

Next, he is given cards each of which bears in printed characters the name of one of the objects in front of him, and he is asked to make his choice.

Both these forms of the test may give him considerable difficulty; and I am therefore accustomed to follow them up by placing a duplicate in the normal hand, out of sight, and asking the patient to choose the corresponding object from amongst those on the table. In all cases of uncomplicated disorders of speech of cerebral origin, this can be carried out perfectly.

He is then asked to write down in turn the name of each object indicated by the observer. This must be carried out in silence; but it is impossible to prevent many patients from moving their lips during the act of writing. After carrying out this test, it is often useful to make the patient name the objects aloud, and then write down what he has said. Occasionally he is asked to copy in cursive handwriting the printed name on the cards, or to repeat each name as it is said by the observer. These two tests are inserted when there is doubt as to the patient's power of repetition and copying.

Let us take as an example the answers to these tests given by No. 2, seven months after the injury to his head. (Table I, p. 95.)

Throughout the first series he never hesitated for a moment, but was prepared to indicate the correct object on the table, even before the screen was removed which hid them from his sight.

With the second series he was much slower. At first his lips moved, forming the word said by me, or he actually whispered or uttered it aloud. He said, "I can't remember what they are unless I think. If it wasn't here on the table I couldn't tell you."

TABLE I.

	(1) Pointing to an object on the table which corresponds to one shown to him	(2) Pointing to an object named verbally by the observer	(3) Pointing to an object named in print	(4) Duplicate placed in his left hand out of sight	(5) Naming an object indicated	(6) Writing the name of an object indicated
Knife ..	Correct and very quick	Correct (whispered "knife")	Correct ; silent	Correct and very quick	Correct..	Wrote "Jan"
Key ..	"	Correct	" "	"	Placed his hand into his pocket, pulled out keys. Said "No, I can't tell you that"	(Gave up)
Penny ..	"	" (repeated "penny")	" "	"	Correct..	Wrote "panin"
Matches ..	"	Correct (repeated "matches")	" "	"	"That's match, mat, mats, match"	Wrote "machin"
Scissors ..	"	Hesitated, moved lips; then correct	" "	"	"That knap, ker, kur-te . . . No, I can't tell you that"	Wrote "naro"
Pencil ..	"	Correct, quick and silent	" "	"	"Pentl, permitch ; not quite it. Pĕ night. No, I can't"	Wrote "knelers"
Key ..	"	Correct ; slow ; silent	" "	"	"That's mer, may".. ..	Wrote "no, karest"
Scissors ..	"	Hesitated ; then correct , silent	" "	"	"No idea. I know exactly what it is, but I can't tell you"	(Gave up)
Matches ..	"	Correct ; quick ; silent	" "	"	"That's match"	Wrote "no merest"
Knife ..	"	Hesitated ; finally correct ; silent	" " slow ..	"	"Nĕ-ife, Ker-nife"	Wrote "knerreg"
Penny ..	"	Correct . slow ; silent	" " very quick	"	Correct	Wrote "pe. penner"
Matches ..	"	Correct ; slow ; silent	" "	"	"That's . . . match" ..	Wrote "mar, matare"
Scissors ..	"	Correct ; quick ; silent	" "	"	"That's . . . No"	(Gave up)
Pencil ..	"	Very slow ; finally correct ; silent	" " very slow	"	"It's a p. something . . . No"	(Gave up)
Penny ..	"	Correct ; quick ; silent	" "	"	Correct..	Wrote "penne"
Knife ..	"	Very slow ; finally correct ; silent	" "	"	"Kĕ-nife. I always remember that . . . It's canif in French"	Wrote "kerrert"
Key ..	"	Correct ; quick ; silent	" "	"	Took his keys out of his pocket and said "Mat, latch"	Wrote "wer, karet"
Matches ..	"	Correct ; quick ; silent	" " quick ..	"	Correct..	Wrote "marost, maters"

When he pointed to an object named in print, the card was held in front of him all the time until he made his choice. His finger hovered in the air, whilst he looked backwards and forwards at the card, until at last he dropped his finger onto the correct object. Except where he made his choice rapidly he reinforced his memory by frequently consulting the printed name on the card in front of him.

When a duplicate was placed in his hand out of sight, he never had a moment's doubt, but indicated the object with great rapidity. Moreover, he remained silent and his lips did not move.

Asked to name an object to which I had pointed, he showed great hesitation and difficulty. His lips moved and he frequently made several attempts in vain to find the word. Occasionally, however, he enunciated it at once, and without preface, but sometimes a correct answer was preceded by "That's . . ."

Throughout his attempts to write the name of an object shown him, he said nothing aloud; but it was obvious from the movements of his lips that he was attempting to find the word with the aid of silent articulatory movements. His power of naming was, however, so bad that it gave him little help in writing the name of the object.

(*b*) *Naming and recognition of colours.*—This test is carried out in the same manner as the one I have just described, except that coloured silks are substituted for the objects of common use. Eight strips of different colours are laid in a row and hidden from the patient by a sheet of cardboard; he is then shown an exact duplicate of one of the pieces on the table and, when the screen is removed, is asked to indicate the one which corresponds with it. Should there be hemianopia or any defect of the visual field, it is important to make sure that the whole gamut of colours is visible to the patient from the position in which he is sitting. Provided this source of error is avoided, the test in this form could be carried out correctly in all the cases of unilateral lesion of the brain comprised in this research.

Then the patient is asked to indicate the colour named orally or in print. Next he says the name of each one in turn or attempts to write it down silently. Lastly, he reads the name of each colour aloud, as it stands on a printed card.

The following example (Table II, p. 97), taken from No. 2, shows many of the most characteristic errors. At first sight it might seem as if he were colour-blind; but this is excluded by the rapidity and correctness with which he matched colours in the first series of observations. The defect was one of names and the gross errors were due to want of

TABLE II.

	(1) Pointing to similar colour to that shown	(2) Pointing to colour named by the observer	(3) Pointing to colour named in print	(4) Naming colour shown	(5) Writing name of colour indicated	(6) Printed name read aloud
Black ..	Correct and rapid	Pointed to white saying "Black"	Chose white ..	"Green—red; no, not red. I can only call it dead."	Wrote "Red" ..	"Red"
Red ..	,,	Correct, whispered "Red"	Chose red very slowly	"Ber-lu. I know what it is. It's what the Staff . . . the same colour I had here. I think it's red"	Wrote "Bell" ..	"Green. No, that's red, blue. No, it isn't blue, it's red"
Blue ..	,,	Correct, whispered "Ber-lu"	Chose violet ..	"Ber-lu"	No response ..	"Green"
Green ..	,,	Correct, whispered "Green"	Chose white ..	Whispered "Green." Compared it with blue band on his arm and rejected it. "I don't know this one. . . . Ber-lu"	Wrote "Gren"	"Green"
Orange ..	,,	Chose yellow, said "O-age." Then chose orange	Chose orange ..	"It's like Pil . . . I think that's red"	Wrote "Read"	"Red"
White ..	,,	Chose yellow silently ..	Chose yellow ..	"That's green"	Wrote "Gernd"	"Red, that's red"
Violet ..	,,	Correct; silent	Chose violet ..	"Ber-lu, ber-loor. . . It's more like mauve"	Wrote "Moved"	"Blue, green; no, mauve"
Yellow ..	,,	"I have no idea. Yellow. I don't know." Finally chose orange	Chose white ..	"That's red"	Wrote "Grena"	"Red"
Red ..	,,	Said "Pred" and chose red	Chose red ..	"What the Staff is again. I can't remember what it is"	Wrote "Blead"	"Red"
White ..	,,	Said "White" and chose white	Chose white ..	"Green"	Wrote "Red" ..	"Green"
Yellow ..	,,	Correct; silent	Chose violet ..	"That is . . . I know what I am trying to remember . . . Kark-too, tark-loon, kar-ki . . that's right"	Wrote "Run" ..	"Red"
Blue ..	,,	Said "Ber-lu; I think it's this one." Very slowly chose blue	Looked at the blue band on his arm, and after great delay chose blue	"Ber-lu"	Wrote "Movy"	"Ber-lu"
Green ..	,,	Said "Dreen, green." Chose white	Chose black ..	Touched his arm, shook his head and said "Mauve. It's not quite mauve, but it's rather like, I think"	Wrote "Gernat"	"Red"
Black ..	,,	Said "Black" and chose correctly	Chose blue ..	"That's red"	Wrote "Mat" ..	"I know this is an ordinary one. Green. No; red, no"
Orange ..	,,	Said "Or-ridge" Chose yellow	Chose orange ..	"I think it's pink"	Wrote "Grend"	"Mauve"
Violet ..	,,	Said "Violet." Very slowly chose violet	Chose violet ..	"Mauve"	Wrote "Moved"	"This one is mauve"

nominal recognition; even the printed names, when read aloud, showed the same striking defects as the choice of the colour to oral command.

(c) *The man, the cat, and the dog test.*—This test is designed to investigate the power of reading and writing in its simplest and most elementary form. Every word employed comprises three letters only; the patient is, therefore, precluded from guessing at the constituents of the phrase by their length, a fallacy which is otherwise liable to vitiate all observations of this class. But, on the other hand, the task set is childishly easy, and the defect in speech must be severe, before it shows traces on so simple a test.

First, the patient is made to read aloud the different combinations from printed cards. Then he is shown pictures of a man, a cat, and a dog in pairs, corresponding to those he previously read aloud. Next, he is asked to write the phrases from pictures or from dictation. Finally, he is made to repeat the words said by the observer, to read what he has written, and to copy from the printed card.

The commonest form of error revealed by this test, as shown on the following table, is a tendency to substitute one name for another. Occasionally this may go so far that the patient inserts "woman" into his answer, oblivious of the fact that no word of this length occurs in the series.

I have chosen for reproduction a set of observations where the six combinations were put once only; it is, however, advisable to repeat them once or more, varying the order each time. (Table III, p. 99.)

(d) *The clock tests.*—Two clock faces are prepared, about twelve centimetres in diameter, fitted with metallic hands, which are adjustable. On each, the figures of the hours are clearly marked in Arabic numerals. One of these clocks is given to the patient, and he is asked to place the hands in an exactly similar position to that of the other one, set by the observer. This direct imitation can be carried out correctly, unless the power of comprehending the nature of the task is considerably diminished.

Next, the patient is told to set the clock to oral and then to printed commands, and in each instance the sequence of tests is the same. The hands of the clock are now moved by the observer into various positions and the patient tells the time aloud, or without speaking writes them on paper.

Most of the characteristic errors discovered by this set of tests can be seen on the following table, compiled from observations on

TABLE III.

	(1) Reading aloud	(2) Reading from pictures	(3) Writing from pictures	(4) Writing from dictation	(5) Repetition	(6) Reading what he has written	(7) Copying
The dog and the cat	Correct; quick	Correct	Correct	Correct	Correct	Correct	Correct
The man and the dog	"The man and the . . . dog" (slow and hesitating)	,,	,,	,,	,,	,,	,,
The cat and the man	Correct; quick	,,	,,	"The cat and the dog"	,,	,,	,,
The cat and the dog	"The cat . . . the cat and the . . . dog" (slow and hesitating)	,,	,,	"The cat and the man"	,,	,,	,,
The dog and the man	Correct; quick	,,	"The cat and the man"	Correct	,,	,,	,,
The man and the cat	Correct; quick	,,	Correct	"The man and the dog"	,,	,,	,,

TABLE IV.

	(1) Direct imitation	(2) Clock set to oral command	(3) Clock set to printed command	(4) Telling the time shown on a clock set by the observer	(5) Writing the time shown on a clock set by the observer
5 minutes to 2	Correct	Set 2.10	Set both hands at 2	Correct	Wrote nothing. Said "I seem to be getting confused between the hands"
Half-past 1	,,	Set 1.10	Set both hands at 1 "I seem to think there is something wrong"	,,	Correct
5 minutes past 8	,,	Both hands set at 8. "I've forgotten"	Set both hands at 8	,,	"Ten minutes past eight"
20 minutes to 4	,,	Both hands set at 4. Then set 4.25	Long hand at 4; short hand at 7	"25 minutes to 4."	Gave up
10 minutes past 7	,,	Long hand at 7; short hand at 5	Correct	"10 minutes past 8 . . . 7"	Correct
20 minutes to 6	,,	Set 6.20	Set 6 15	"25 to 6. . . I seem to get in a maze, when I go back to it again"	"Ten minutes past seven"
10 minutes to 1	,,	Set 1.10	Set both hands at 2	"5 to 8. No, that's 10"	Correct
A quarter to 9	,,	Correct	Correct	Correct	"Quarter to eight"
20 minutes past 11	,,	Long hand at 11; short hand at 10	Long hand at 11, "I've no certain opinion. It will be a case of tossing up"	"10 minutes past 11. . . I get confused between the long hand and hour hand"	"Ten minutes past four"
25 minutes to 3	,,	Shook his head. Set long hand at 3. Then gave up	Set 3.35	"Quarter past 7"	"Half past seven"

No. 10. (Table IV, p. 100.) The patient mistook "to" and "past" the hour, or confused the two hands; thus he set the long hand at the hour and either placed the short hand in some erroneous position, or became confused and gave up the attempt. Except when he was imitating the position of the hands on the second clock set by the observer, he usually placed the short hand exactly on the number of the hour. For example, when asked to set "twenty minutes to four," he placed it opposite four, so that when the minute hand was brought to seven, the ordinary reading would appear to be 4.35. This inability to divide the space between the figures of the hours led to much difficulty in recording the position into which the hands had been placed, unless that assumed by each one was noted separately.

(*e*) *The coin-bowl test.*—Four bowls or saucers are set upon the table and in front of each is laid a penny. The patient is told to count them from left to right; he is then shown the nature of the task he has to perform, which consists in placing a coin into one of the bowls according to a series of numerical commands. First, the order is given orally, or by means of a printed card, which is read silently. Then the patient is asked to read it aloud, and to carry out the action demanded under the influence of words spoken by himself.

This is so simple a test that no normal person fails to carry it out correctly; but in some instances of aphasia and kindred disorders it may cause considerable difficulty, as seen by the example given on table. In this case both verbal and printed commands were poorly executed, although when asked to read the order aloud he did so correctly. (Table V, p. 102.)

It is sometimes useful to make two sets of observations with the printed cards under somewhat different conditions. First, the orders are given in numerals only, e.g., "1st into 3rd"; then the whole phrase is set out in full, as for example, "First penny into third bowl." In cases of aphasia the second form of this test usually presents greater difficulty to the patient, and may reveal defects that do not appear when the command is given in numbers only.

(*f*) *The hand, eye and ear tests.*—First of all the patient, seated opposite the observer, attempts to imitate a series of movements which consist in touching an eye or an ear with one or other hand. Before beginning these tests it is well to make sure that he knows his right hand from his left, and understands the nature of the task he has to perform. He is asked, therefore, to name each hand as it is raised by the observer, and then to lift the one which corresponds

TABLE V.

	(1) Oral command	(2) Printed command (not read aloud)	(3) Printed command read aloud	
			(a) Said	(b) Action performed
Second into third	Slow; correct	Correct	"Two . . . Three"	Correct
First into third	Hesitated; finally correct	Very slow; much hesitation; correct	"One . . . Three"	"
Second into first	Second coin . . . "I forget which one." Order repeated; quickly correct	First coin . . . Corrected to second coin. No further response	"Two . . . One"	First coin . . . into first bowl, saying "One, one"
Third into second	Third coin . . . "I forget which it was." Order repeated; correct	Second coin into third bowl	"Three . . Two"	Third coin . . . into third bowl
First into fourth	First coin . . . No further response. Order repeated; correct	First coin . . . After two minutes' hesitation, correct	"One . . . Four"	First coin . . . "Was it four?" pointing to fourth bowl. "I forget." Did nothing further
Fourth into third	Third coin into second bowl	Third coin into fourth bowl	"Four . . One . . Three"	Third coin into third bowl
Second into fourth	Correct	Correct	"Two . . . Four"	Correct
Fourth into first	Fourth coin . . . "I forget which one." Order repeated; correct	Correct	"Four . . . One"	First coin. "Four" . . . into fourth bowl
Third into first	Correct	Third coin . . . "I forget which one I've got"	"Three . . One . . Three"	Correct
First into second	Second coin to first bowl; quickly	Correct	"One . . . Two . . . Two"	Correct. "Was that it? I don't know"
Third into fourth	Extremely slow but correct	Third coin . . . "I forget which one to put in. It's one of these," pointing to third and fourth bowl	"Three . . . Four"	Third coin into third bowl. . . . Removed it and replaced it in third bowl
Fourth into second	"I forget which it was." Order repeated: fourth coin into first bowl	Correct	"Four . . . Two"	Correct

to it, bearing in mind the face to face position. When we are certain from these preliminaries that he appreciates the action demanded of him, the observations are begun.

Some normal persons find difficulty in performing these movements correctly over a series of from sixteen to twenty-four tests; many, however, can carry them out perfectly, especially if they are young and intelligent, and belong to the class from which so many of my war patients were drawn. There is a natural tendency to select the hand opposite to that used by the observer; this error is in most instances checked consciously. But none of the normal men I have examined failed to recognize that when the left hand was in contact with the right ear it had crossed the face; and yet this want of appreciation of crossed movement was one of the commonest pathological mistakes. Moreover, in certain cases, I have been able to watch a steady improvement in the records, resulting finally, after a considerable lapse of time, in a perfect series of answers.

Then the patient is placed in front of a large mirror, and is asked to imitate the reflected movements of the observer standing behind him. In all normal persons and in most of those suffering from disorders of speech, this can be carried out perfectly; it is an act of direct imitation uncomplicated by considerations of right and left, and not attended by the necessity for internal speech.

The next form of this test consists in handing the patient cards, each of which represents a human figure carrying out one of the desired movements. These drawings are simplified to the highest degree consistent with their significance, and a line is drawn down the centre to separate the right and left halves of the body. Most patients, when shown these pictures, make exactly the same kind of mistakes as when seated opposite the observer. But as soon as they are allowed to see the reflection in the glass, every movement may be executed rapidly and correctly.

Then the patient is made to carry out the same series of actions in response to oral and to printed commands. He is next asked to read aloud each order, and to execute it under the reinforcing influence of words said by himself. Finally, he is asked to write down in silence the movements made by the observer sitting opposite him. Occasionally, where the power of writing is in question, he is asked to copy the orders printed on the cards.

Table VI, p. 104, illustrating these tests, was obtained from observations on No. 4, a case of so-called "motor aphasia."

TABLE VI.

	(1) Imitation of movements made by the observer	(2) Imitation of movements of the observer seen in the glass	(3) Carrying out pictorial commands	(4) Pictorial commands seen in the glass	(5) Oral commands	(6) Printed commands (not read aloud)	(7) Reading aloud and executing printed commands	(8) Writing down movements made by the observer
Left hand to left eye	Correct	Correct	Right hand to right eye	Correct	Correct	Correct	Correct	"Left left eye"; correct
Right hand to right ear	Left hand; then right hand; correct	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand right ear"; correct
Right hand to left eye	Correct	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left eye"; correct
Left hand to right eye	Left hand to left eye ..	,,	Right hand to left eye	,,	,,	,,	,,	"Left hand, right eye"; correct
Left hand to left ear	Correct	,,	Correct	,,	,,	,,	,,	"Left hand" ("I forget which it was")
Right hand to right eye	Correct	,,	Left hand to left eye	,,	,,	,,	,,	"Right hand right eye"; correct
Left hand to right ear	Left hand to left ear; "I have to think"	,,	Right hand to left ear	,,	,,	,,	,,	"Left hand right ear"; correct
Right hand to left ear	Right hand to right ear	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left ear"; correct
Left hand to left eye	Right hand; then correct	,,	Correct	,,	,,	,,	,,	"Left hand left eye"; correct
Right hand to right ear	Correct	,,	Left hand to left ear	,,	,,	,,	,,	"Right hand" ("I think it was the same side")
Right hand to left eye	Right hand to right eye	,,	Left hand to right eye	,,	,,	,,	,,	"Right hand left eye"; correct
Left hand to right eye	Right hand to right eye; then right hand to left eye; "I see the mistake"	,,	Right hand to left eye	,,	,,	,,	,,	"Left hand to right eye"; correct
Left hand to left ear	Correct	,,	Correct	,,	,,	,,	,,	Wrote nothing ("I've forgotten")
Right hand to right eye	Correct	,,	Correct	,,	,,	,,	,,	"Right hand right eye"; correct
Left hand to right ear	Correct	,,	Right hand to left ear	,,	,,	,,	,,	"Left hand right eye"
Right hand to left ear	Left hand to left ear ..	,,	Left hand to right ear	,,	,,	,,	,,	"Right hand left ear"; correct

This is the most difficult of all the serial tests, and the only one where the answers of a normal person may be at fault. But, if we bear this possibility in mind, it is capable of giving valuable information concerning the nature of the different defects of speech of cerebral origin.

§ 2.—*Further Tests employed in this Research.*

It is customary to test a patient, who suffers from one of these disorders of speech, with the alphabet and, if he shows any difficulty in naming the letters, he is given a pencil and told to write them in their proper order. But this is not sufficient to determine his disability; his capacity to carry out this test must be examined systematically. He is told to say aloud, to write and to read the alphabet; then he takes it down from dictation, copies it from printed capitals in cursive handwriting, and finally, if the formation of the sounds is defective, he is asked to repeat it letter by letter after the observer. Occasionally he is given the letters of the alphabet printed on separate cards, and is asked to arrange them in order.

A similar set of observations are carried out, using as a basis the days of the week or the months of the year.

Another valuable method of examination belonging to this order is to choose some paragraph in the newspaper of interest to the patient, and then to ask him to retail the information in different ways. For example, he is allowed to select an account of the latest boxing match, and asked to read it to himself silently; after the paper has been removed he narrates what he has gathered, and then writes down his own account of the fight. Then he is asked to write it from dictation, to copy the printed account in handwriting, and to repeat it verbally; phrase by phrase, after the observer. Finally, he attempts to read aloud what he has written, however defective it may be. The order of these operations is determined by the class of patient with whom we have to deal, and the nature of his defect. It is extremely important, however, that the task selected should be of interest to him, and it must not be long or difficult to remember; moreover, it should be strictly adapted to the extent of his education.

It is customary in these cases to place a picture before the patient, and to ask him to describe in spoken or written words what he sees. Sometimes a combination of this pictorial test with the comprehension of printed matter in the following way makes a useful method of examination. A picture is chosen to which is attached a short printed description, so common in the daily press. At first the legend is covered up; the patient is asked to say what he sees in the picture, and the manner and nature of his observations are carefully recorded. Then he is allowed to see the printed description; after reading it silently to himself he writes down what it conveys to him, reinforced by the pictorial representation. He then reads it aloud and writes it to dictation. Finally, he is told to fix his attention on the picture, and to say what it conveys to him. An excellent example of this combined test is given on p. 139.

To be of any value arithmetical tests must be simple and carried out systematically in the order of their progressive difficulty. I am in the habit of beginning by asking the patient to add two simple numbers of three figures each, such as 235 and 462, which do not necessitate carrying over. This is followed by a couple which require one act of carrying over, and then by another where it is necessary to perform this operation twice.

From this he passes to the subtraction of one number of three figures from another, where no carrying over is required; the difficulties are then increased progressively in exactly the same manner as with addition. All these tests are absurdly simple to the normal man, provided he is not illiterate.

Great stress will be laid on the patient's ability to play games, such as chess, draughts, cards and billiards. "Jigsaw" and other puzzles may also give valuable indications of his aptitudes and disabilities.

His power of drawing must be tested in a more systematic manner than is usually the case. First, he is asked to draw from a simple model, such as a bottle or a vase. If he is fond of drawing and naturally intelligent, I sometimes put before him a glass spirit-lamp, because of the difficulties presented by the wick; for the parts above and below the collar must be shown as a continuous structure interrupted by the metallic neck of the lamp. This forms a useful problem in significance. Then both the model and drawing are removed, and he is asked to reproduce it from memory.

Another valuable test, which has been used by Marie and others, is to ask the patient to draw an elephant. Fortunately most of my patients were familiar with the appearance of these animals, and many had seen them in India during their military service. If possible, the patient should also be induced to draw images which come into his mind spontaneously, apart from any external command.

He is also asked to sketch roughly a ground plan of some room with which he is familiar, such as his ward in hospital. In some instances this is carried out with ease, whilst in others it is entirely impossible. But although the patient may be unable to put down on paper any of the significant objects in the room, his visual images are not of necessity defective. For if I draw an oblong in the centre of the paper to represent his bed, he may be able to indicate to me the position of the windows, doors, furniture, and other appurtenances of the ward with absolute correctness. He may know their position, but be unable to represent their relation to one another.

So far, all the tests I have described are new or, if commonly used, are employed by me in an unusual manner. They are not in any way complete, and are capable of profound improvement; but I have set them out in detail to show what methods have actually been employed in this research.

CHAPTER II.—PREVIOUS EXPLANATIONS OF APHASIA.

Gross injury confined to one cerebral hemisphere can disturb the power of speaking, reading, and writing, without producing any other severe loss of intellectual capacity. Evidently there must exist a group of functions indispensable for language in its widest sense, but not equally essential for all intellectual performance; it is these functions which form the subject of this investigation in as far as they can be affected by destruction of certain parts of the brain.

An organic lesion can only cause a disorder of speech by disturbing underlying physiological processes; the structural changes prevent the orderly performance of certain cortical functions, and speech suffers in consequence of a disturbance of vital processes, which may never directly influence consciousness. These abnormal reactions are apparent to the external world in terms of a disorder of language, just as some defect in the mechanism of a clock is manifested by failure to keep correct time. The physiological activities of certain portions of the cerebral cortex are responsible for the capacity to make use of language in a normal manner; when they are imperfectly carried out, from whatever cause, the acts of speaking, reading, and writing become more or less disordered.

Before we can determine anatomically what parts of the cortex are responsible for these manifestations, it is essential to discover the nature of the disorder itself. Unfortunately the actual phenomena of disordered speech have been dismissed for the most part in a very summary manner, although an immense amount of time and energy has been expended on the distribution of the structural changes. Clinicians assumed that the defects in the use of language corresponded to three more or less independent groups, loss of speaking, reading, and writing; these in turn were attributed to destruction of "motor," "auditory," and "visual" images. On the other hand, to Marie and his followers loss of speech is in reality a high-grade change in the mechanism of articulation, whilst all other "aphasic" manifestations are attributed by them to a lowering of general intellectual capacity.

This chapter is devoted to considering in how far the actual nature of these disorders of language corresponds to the categories generally laid down. I shall attempt to show that they cannot be classed under the headings of speaking, reading or writing; for, whenever the structural changes affect one half of the brain only, disorder of one of these "faculties" is accompanied by some analogous change in the others. Then I shall bring evidence to show that such affections of language are not based on a destruction of "images." They obey the law laid down by Jackson fifty years ago, that the negative manifestations of a lesion are expressed in some disorder of the affected function; in this instance they appear in terms of language in its widest sense, and not as a disturbance of images, "motor," "visual" or "auditory."

Finally, I shall examine the suggestion that the "motor" aspect of aphasia is in reality an "anarthria," and shall bring evidence to show that this is not an adequate explanation. But the full consideration of the second half of Marie's theory must be postponed to a later period, when I consider more fully the effect on general intellectual capacity of the disorders of language which I am about to describe.

§ 1.—*Disorders in the use of Language, due to an Unilateral Lesion of the Brain, cannot be classed under the Categories of Speaking, Reading and Writing.*

Had it not been for the trend of scientific thought at the time when the facts of "aphasia" were discovered, no one would have imagined that a lesion of the brain could affect exclusively such complex acts as speaking, reading and writing. But it was assumed that man had been endowed at his creation with certain "faculties," situated in different parts of his brain, and these were thought to be disturbed by the cerebral injury.

This theory seemed to receive support from the fragmentary and insufficient examination of the earlier observers. The patient was said to be able to read and write although he could not speak. Sometimes, on the other hand, he could speak but could not write. But as observations accumulated and the first novelty of the discovery wore off, it became obvious that pure instances of "aphasia," "alexia" or "agraphia" must be extremely rare, and most patients were said to show a "mixed affection" of speech.

From 1866 onwards Jackson opposed such conceptions, and laid stress on the importance of recognizing that there was no such thing as a "faculty" of language. In the same way he insisted that there is no "faculty of memory" apart from the things remembered. But although the doctrine of human faculties may have passed out of fashion, the ideas upon which it was based are still enshrined in the terms "aphasia," "alexia" and "agraphia." Many still hold that it is possible to say that a man has been deprived of the power to speak, to read, or to write by a cerebral lesion, without specifying the conditions under which these complex activities are impossible. It is therefore worth while to demonstrate how little such terms correspond to the actual clinical phenomena.

These "speechless" patients are not "wordless," for they can swear or even ejaculate appropriately on occasion; a man usually speechless may at times produce a complete phrase. But the words of a speechless patient are not at his disposal for voluntary use; they exist for comprehension and can be called up under emotional stress. Voluntary power is diminished with retention of the capacity to evoke the same movement in a more automatic manner. A patient who was unable to find the name for "ink" finally said, "That's what I should call a china pot to hold ink." The disturbance of voluntary speech had destroyed the power of finding the more selective answer "ink," but permitted a more descriptive response. In considering the power to use words we cannot say that the patient is totally speechless; we have to determine under what conditions speech is or is not possible.

In the same way a man may be apparently unable to read a single word, but when a series of printed names of colours are placed on the table, he may be able to point to the one which corresponds to a coloured object in front of him.

The majority of those who are said to be suffering from "agraphia" can write their names and addresses. One patient under my care wrote without difficulty his name, followed by the full address of his home including the county, but he could not do the same for his mother with whom he lived. In this case the words could be written, when they applied to himself and were more nearly automatic, but not in connexion with another person.

We are face to face with exactly the same difficulties when attempting to determine the power of appreciating spoken words. What, for example, is the condition of a man who cannot draw a "square" when asked to do so, but told to draw a "block of wood" at once draws a perfect square?

Obviously, therefore, we have no right to be satisfied with the statement that the patient cannot speak, read or write. It is our business to discover the conditions under which he can or cannot perform any of these acts. As soon as we examine the clinical phenomena from this point of view, we find that no one of these three categories of language is affected alone by any unilateral lesion of the cerebrum. A disturbance of one aspect of speech is associated with some other disorder in the use of verbal symbols.

This is true even of the form usually called "motor aphasia." For although at first sight it may seem as if the difficulty was confined to the act of speaking, closer observation shows that other functions are also disturbed. For example, in No. 17, writing revealed the same kind of defects as speech; even when well on towards a full vocabulary he addressed a letter to his "Bank Man-ger" and, when silently recording the names of geometrical figures, wrote "pymared" and "pymerad," an exact reproduction of his method of pronouncing these words.

Jackson pointed out the close association between speaking and writing, insisting on their coincident variation.. He thought of internal speech as words that had not passed over the vocal organs, and he used writing as a means of testing its condition. Now, internal speech in this sense plays an essential part in many other acts besides speaking and writing. When the patient, sitting opposite to me, attempts to imitate movements of my right or left hand brought into contact with one or other eye or ear, internal verbalization occurs as a phase of the normal act. No word may be uttered, but the words "right" and "left," "eye" and "ear" are essential to correct imitation of this kind. Such imitative acts may, therefore, suffer profoundly in cases of so-called "motor aphasia" [*vide* Table VI, p. 104]. For the same reason these patients may find considerable difficulty in carrying out the hand, eye and ear tests, when the command is pictorial, although they can execute them correctly when asked to do so by word of mouth. Their difficulty is in evoking the words they require, and verbal command supplies the necessary want.

Much has been made in the past of "agraphia" as a nosological entity. But, provided the lesion of the brain is unilateral, inability to write is always associated with some other loss of function. This is particularly evident when the power of naming is defective; the patient not only finds difficulty in writing down the time as part of the clock test, but he also fails to set the hands correctly to verbal or to printed command. Even in those cases where word formation, naming and reading aloud can be carried out perfectly, writing may suffer badly, and the patient may be unable to execute verbal, printed or pictorial commands.

Thus it is impossible to classify the disorders of language produced by a unilateral lesion of the brain as exclusive affections of speech, reading or writing. For this reason, such words as "aphasia," "alexia," and "agraphia" should never be employed to record the actual phenomena in any instance of speech defect.

§ 2.—*These Disorders of Language are not due to Destruction of Images.*

In 1879, at a time when everyone assumed that destruction of auditory and visual images was a sufficient explanation of the phenomena of aphasia, Jackson recognized that images, together with those unconscious processes on which they depend, might remain intact in the speechless patient. He cannot speak, he cannot write, he cannot read, not because he has lost "images" or "memories" of words, but because he has lost the use of words in speech. In cases where images are disordered, there is a peculiar defect which Jackson called "imperception." Speechlessness may exist without imperception, and it is from such instances only that we can obtain a clear analysis of the processes which make up speech.[1]

I could bring forward innumerable examples from my records of the truth of these statements. In fact, it is difficult to understand how any observer who had studied patients with these disorders of speech or had listened to their own account of their disability, could ever have supposed that "images" were destroyed. They repeatedly insist that they can "see" the dog you ask them to draw, or the room they are attempting to describe. Moreover, most of them have little difficulty in drawing from memory an object that has been shown to them and then removed from sight.

---

[1] I hope to consider in a further communication the clinical manifestations of "imperception" and "mind-blindness."

This dissociation between visual images and those activities of language which have usually been attributed to them is well shown by asking the patient to draw a rough plan from memory of some room, such as his hospital ward, with which he is familiar. In many cases, he is entirely unable to do so. But if the observer places a mark on a sheet of paper to indicate the bed on which the patient habitually lies he cannot infrequently indicate correctly the site of windows, doors and fireplace, together with the position of the familiar objects by which he is surrounded. Visual images are intact, but they cannot be represented in the conventional manner. In the same way, No. 2 produced to command the most fantastic and absurd representation of an elephant, although his spontaneous drawings of animals were remarkably good. Images were present but they could not be reproduced to order. The same patient could play an excellent game of chess, which in his case seemed to depend on a strong power of visualization; cards, however, puzzled him and he was quite unable to score.

Auditory images were supposed to be responsible for "memories" of words and they were said to be "stored up" in certain areas of the cortex. But this hypothesis is entirely incapable of explaining the phenomena of aphasia. An officer, who in consequence of a bullet wound of the head had lost the power of speaking, reading and writing, kept beside his bed a list of his possible wants. He would ring his bell and, when the nurse arrived, point without fail to the name of the object he required.

The elderly man, whose condition is described on p. 115, could not speak, read or write. If a card bearing the name of a colour was placed in his hand he was unable to read it: the letter conveyed nothing to him. In the language of the older theory he had "lost the memory for words." But if eight cards, each bearing the name of a colour, were placed on the table in front of him, he could pick out the one which corresponded to an actual colour shown to him.

It is useless further to labour this point. For there is not a single manifestation presented by the defects of language, due to a unilateral lesion of the brain, that can be explained by destruction of auditory or visual images. The theory had its birth in the study and is contrary to the facts of clinical observation.

§ 3.—*The "Motor" Aspect of these Disorders of Language is not a pure "Anarthria."*

In 1906, Pierre Marie broke away from all the orthodox views of aphasia and propounded the theory that the "motor" aspect of disorders of speech was due to "anarthria," a high-grade disturbance of articulation. On the other hand, he held that the "aphasic" aspect was nothing more than a manifestation of diminished general intelligence. "Il n'y a rien d'aphasique," he says, "dans le trouble *moteur* de l'anarthrique. L'anarthrique comprend, lit, écrit. Sa pensée est intacte, et l'expression en est possible par tout autre moyen que la parole, le langage intérieur n'étant pas altéré."

This statement had the advantage of clearing the ground of older preconceptions, but unfortunately substituted a theory which does not agree with the results of more extensive clinical observation. Marie contends that the anarthric patient, in his sense of the word (or the "motor aphasic" as it was the custom to call him), understands, reads, and writes; his thought is intact, he can express himself by every other means except words, and internal speech is unaffected.

Now it is true that such patients may be able to read and write; but the statement of Marie and his followers implies that these acts can be performed perfectly, and that emissive speech is alone disturbed as a consequence of the brain lesion. Closer observation shows that this is not strictly correct. Amongst my military patients were three who would have been spoken of as uncomplicated examples of "motor aphasia"; they could read and write, and showed no defect of general intelligence or memory, and would so far correspond to Marie's definition of anarthria.

Further investigation, however, showed that other functions were affected besides emissive speech, and this was borne out by the statements of the patients themselves. No. 17 talked slowly and was frequently at a loss for a word; he said: "I sometimes — — have to — — alter the whole — — to alter the sentence — — because — — I — — have — — difficulty — — in finding — — the word." Often he substituted some more picturesque expression, such as "dig it up" for "remember." "I want to-say a word — — at the back of my mind — — I can't just — — dig it up." He added, "At first — — I — — don't think — — I'd more — — than a twenty-word — — voc — — abulary." When saying the alphabet aloud he stumbled over "R"; this he explained by saying that, when he had trouble over "R," it was not that he did not know it, but he wanted to say "A" instead of "R," because he was thinking of "are." If he was not certain of a thing he wrote it down; "I may write it wrong — —, but when I see it — — I know it's — — wrong."

He recognized that he frequently found difficulty with adjectives. "I said a person was strong-willed and strong-headed, and I knew it was not what I wanted to say; half a day afterwards I said, 'You wanted to say headstrong.'"

He could understand what he read if he went slowly, and he never misinterpreted the meaning. But he confessed: "I can't read a book to myself because I'm bothered when I say the words. I can get the meaning of a sentence if it's an isolated sentence, but I can't get all the words. I can't get the middle of the paragraph; I have to go back and start from the preceding full-stop again."

When asked to read a few lines from a book to himself and to write shortly what he had remembered, he did so slowly and with great effort. Moreover, his errors were exactly of the same order as those in speaking. He explained that though he could write he did so with difficulty: "I always have to spell out every word, even the little ones. I have to say 'of'; I know it's a preposition, but then I have to think, Is it 'to' or 'from' or 'of'? Prepositions are always a bother to me."

He was shown a set of geometrical figures and named them correctly, although his pronunciation was defective; he was then asked to write down their names without speaking, and although he never failed to indicate their shape, he wrote "pymared" and "pymerad" instead of pyramid. This closely corresponded to the sort of mistakes he made in pronunciation. Another highly intelligent officer described his disability as follows: "I'm confine (confined) to the words I've got back since my speech came back. I try to make a statement—I have to use the words I've got back—when they say, 'What do you mean by so and so?' I haven't got a further example of what I've said, to explain. My vocabulary (pronounced 'vocab-lery') is still small. When I try to explain I haven't got the words I want, and when they say over a set of words, I then say 'Yes, that's the one.' I have to go over the roots; I trace the root back to the Latin or that, and get it eventually (pronounced e-venchaly)."

Even stronger evidence, that this condition is something more than an uncomplicated disturbance of emissive speech, is given by the hand, eye and ear tests. The patient sits opposite to the observer and imitates his movements exactly; in this class of case, the task is carried out slowly, and with a varying proportion of mistakes. But as soon as the observer stands behind the patient, and the movements are reflected in a mirror, they are executed perfectly and with great rapidity. An exactly similar phenomenon occurs when the order is given by means of a series of pictures; the patient has much difficulty in executing the movements when he holds the card in his hand, but none when it is reflected in the glass. No. 17 explained his difficulty as follows: "I've always said it is like translating a foreign language, which I knew but not very well. It's like translating from French into English." This patient entirely recovered his speech except for a certain hesitation, and three years later carried out this test in all its forms, without the slightest difficulty.

All these facts and many more, which will be brought forward as examples of the nature of the disturbance produced by these unilateral lesions of the brain, show how little the "motor" disorder of speech can be explained as an uncomplicated "anarthria."

CHAPTER III.—THE NATURE OF THE FUNCTIONS DISTURBED IN APHASIA AND KINDRED DISORDERS OF SPEECH.

NO conception that we have so far considered is in harmony with the clinical manifestations of these disorders of language: for any satisfactory hypothesis must be capable of explaining why the patient succeeds in reading or writing under certain conditions, although he fails completely if the task is presented to him in a different manner.

Let us then first of all consider, from this point of view, an ordinary case of so-called "aphasia" with "alexia" and "agraphia." On the evening of May 13, 1911, a man of 56 suddenly lost all power of speaking naturally; next day he was drowsy and the disorder of speech increased. On May 17, he came to my out-patient department, and was admitted to the wards, suffering from severe aphasia without hemiplegia. He was discharged a month later, and remained constantly under the care of Dr. Percy Kidd, until his sudden death on January 6, 1920. His condition throughout this period of nearly nine years remained unchanged; but I was able to make an extended series of observations in November, 1919, and it is on these that the following conclusions are based.

He was almost completely speechless, but could reply "Yes" and "No" correctly: asked to say "Yes," he answered "No, I can't" and told to repeat "No" he usually shook his head, but once said "No, I don't know how to do it." He could at times use the expression "Thank you" appropriately, and when puzzled would say "Can't get it; I know what it is, I try." Once when excited by the request to draw an elephant, he said: "Yes, I've been all over; seen lots! I've got some on my what you call it," alluding to figures on his sideboard at home.

He could not name a single object or tell the time set for him on the clock. But when the hands were placed at 1.30, he held up one finger and bisected it with the other hand; shown 1.55, he held up five fingers and then two, to indicate five minutes to two.

He carried out oral commands excessively badly. Even with the coin-bowl test, he failed in twelve out of fifteen attempts to place the penny named in the right bowl; and yet he had no difficulty in pointing to any one of a series of objects, or choosing the correct colour named to him verbally. It was not words that were lacking, but words used in a certain manner.

Asked to imitate my movements, when I touched the right or left eye or ear with my right or left hand, he failed grossly, sitting face to face. But when my actions were reflected in the glass, he never made a mistake and did not even hesitate. An exactly similar result followed on pictorial commands of the same kind; holding the card in his hand, he was wrong every time, but with the picture reflected in the glass, he carried out the movements perfectly and with great rapidity.

The most remarkable incongruity was shown, however, when he attempted to read. Given a printed command, such as to touch eye or ear with one or other hand, he made no effort to carry out even the slightest movement. He shook his head, saying, "I can't, I know what it is, I don't know what it says." In the same way with the coin-bowl test, he did not attempt to carry out a command given either in printed words or numbers.

But in spite of his inability to execute printed commands, there was no doubt that he could read. He was able to point correctly, on seventeen out of eighteen consecutive occasions, to an object named in print. This exactly corresponded to the results obtained when they were named orally. He was able to carry out an even more severe test on similar lines. The six printed cards of the man, cat and dog test were laid on the table in front of him. Then he was shown pictures of one pair of the series, for instance, the man and the dog. He picked out correctly, ten times in succession, the card which corresponded to the combination he had seen. Once he chose a card with the inscription, "The cat and the man," but rejected it for one bearing "The man and the cat," the order from left to right in which the figures had been presented to him. Later in the same series, he again chose a card with the right names in the reverse order, but immediately corrected his mistake. Thus, he was not only accurate with regard to the verbal significance of the card he selected, but also paid attention to the order of the words in print.

His signature was reduced to one word, "Beaden" (Beaton), and he was totally unable to write the alphabet when asked to do so; but if it was dictated, seventeen of the letters were perfectly formed and many of the remainder were recognizable.

He could not name a single coin, but indicated the relative value of any two of them by holding up the exact number of fingers. Thus, when a penny and a shilling were laid on the table, he raised his hand with all five fingers extended; said "Nover" (another), held up his hand again and then added two more fingers, making twelve in all. He could even give the relation between a penny and a half-crown, raising his hand six times in succession: and yet he could not count above nineteen, and was unable to carry out simple addition or subtraction.

Asked to draw an elephant, he produced an absurd figure bearing no relation to any living creature. He marked in what appeared to be an eye; asked what it was, he said, "Don't know" but pointed to his own eye. Questioned about a peculiar horn-like projection on the front of the drawing, he caught hold of his own ear; and when I asked "What is that?" pointing to the object hanging down in front, he passed his hand over his own face, moving it upwards, saying, "Sometimes up that way." This was evidently intended to represent a trunk.

It is obvious that this patient had not lost the power of reasoning and was in many ways highly intelligent. Although he was almost entirely deprived of speech and could not name anything placed before him, he could indicate the object or colour named to him orally or in print. He imitated movements extremely badly, and if I sat opposite him or if the order was given in the form of a picture; when however such commands were reflected in the glass, they were carried out perfectly. For, in the first case, the words right and left, eye and ear, must be silently interposed between the reception and execution of the command; but when reflected in the glass the movements are purely imitative and no verbalization is necessary. When shown a common object of daily use or a colour, the patient had no difficulty in pointing to the corresponding one amongst a group on the table before him. Immediate or intuitive recognition caused him no difficulty.

What then is the function which is disturbed by such lesions of the brain? It obviously cannot be comprised under the headings of speaking, reading and writing; for not only may the loss of power to carry out any one of them be partial, but the disturbance extends beyond their limits and affects other mental activities.

All patients suffering from the disorders dealt with in this paper can choose from amongst a set of common shapes and colours the one which corresponds to that which they have been shown. If a simple geometrical figure or one of a series of objects in everyday use be placed in his normal hand out of sight, the patient can indicate without hesitation the similar one from a group in front of him. He is able to set one clock in imitation of another without difficulty. He can, in fact carry out any operation not demanding symbolic formulation. All forms of immediate recognition are possible which do not require the intervention of some symbol; but all acts of symbolic thinking and expression are liable to be affected, more or less, in the cases of disordered language.

Words are the commonest and most obvious symbols used in thinking, and any action is liable to suffer which demands for its perfect execution any form of verbalization. A gesture which can be accurately imitated, when reflected in the glass, cannot be performed with certainty if patient and observer are seated face to face; for this attitude necessitates translation of the movement into words before it can be carried out. If, however, the command is given in spoken or printed words, it may be executed correctly, because the necessary symbols are provided in the order itself. The patient is not compelled to formulate them, as when he attempts to translate into action gestures made in the face to face position or their pictorial representations.

Any act, demanding for its correct execution a formulated proposition, will certainly be affected, whilst the more closely it corresponds to the matching of two patterns the less it will suffer from these disorders.

This conception almost exactly corresponds to the views put forward by Hughlings Jackson from 1868 onwards. He stated that the words removed in consequence of unilateral lesions of the brain were those employed in the "formation of propositions"; those which remain to the "speechless" patient are the same words used "non-propositionally" or in the lowliest form of "proposition." Less severe destruction of speech disturbs the use of words in such a way, that the higher and more abstract the "proposition" the more likely is the patient to fail, not only in the emission of a correct verbal equivalent, but in the recognition within himself of the full value of the "proposition." As Jackson expounded this theory in paper after paper, it assumed a form which includes the greater number of the facts I have observed.

It is with the greatest reluctance, therefore, that I venture to change his nomenclature; for I believe that under the uncouth word "propositionizing" is included what I understand by "symbolic thinking." This, Jackson contrasted habitually with what he called "lower forms" of speech or thought. But the question as to what constitutes a proposition is so disputable that it is better to avoid a term which is liable to be misunderstood, and to lead to controversy. Moreover, it is doubtful whether the term is strictly accurate even in Jackson's sense; and it certainly does not cover all the abnormalities observed in cases of aphasia and kindred disorders.

I would therefore suggest that the function affected in those pathological conditions might be called "symbolic thinking and expression." The following chapter is devoted to the various forms assumed by the clinical manifestations, and, after a description of these abnormal states, it will be possible to enter more fully into the nature and limits of this disordered function.

CHAPTER IV.—DISSOCIATED FORMS OF SYMBOLIC THINKING AND EXPRESSION.

In the previous chapter I have suggested that the various disorders of language produced by a unilateral lesion of the brain might be grouped together as affections of symbolic thinking. Every manifestation of this aspect of psychical activity is not, however, of necessity disturbed in any one case: but the more acute and severe the lesion, the graver and more extensive will be the disorder it produces. Sometimes all the specific tests yield an abnormal result; in other instances, if the injury is slighter and more strictly local, many of them may remain unaffected. This leads clinically to the appearance of those dissociated manifestations which gave rise to the conception of various forms of aphasia.

But the consequences of this dissociation are expressed in terms of the function itself, and not as a destruction of auditory and visual images or other processes, which do not form an integral part of the act of symbolic thinking. They belong to the same level of psychical activity, and under normal conditions form correlated factors in the production and comprehension of language in its widest sense. Each of them, however, requires the orderly performance of certain physiological processes, which are disturbed by the destructive or paralysing effect of the lesion. These are not all dependent on the functional activity of the same region of the cortex, and consequently may be affected more or less independently. Each aspect of symbolic thinking is associated more closely with the physiological life of certain parts of the brain, and a local lesion can therefore produce a more or less partial disturbance. The nature of these various defects forms the subject of the present chapter.

But before passing on to describe the actual clinical phenomena, I must utter a warning against the erection of new "types" of aphasia. All the disorders described in this paper are affections of symbolic thinking, and it is this psychical function which is disturbed in consequence of the organic lesion. An exactly analogous dissociation may accompany a lesion of the so-called sensory cortex. All the physiological activities associated with the discriminative aspects of sensation are not uniformly distributed within this area, and a local injury, provided it is not severe, can affect some and not others. The sensory loss is partial; certain tests are affected whilst others give normal results. Such dissociation does not express the elements out of which sensation is primarily composed, but reveals the various forms into which this complex act can be broken up in man. It shows that, with the gradual development of the highest strata of the nervous system, certain parts became more peculiarly associated with the physiological processes underlying particular aspects of sensation. Thus, injury to the sensory area of the cortex may produce complete loss of all sensory discrimination on some portion of the body, or the disturbance of sensibility may be partial, affecting one aspect more than another.

The defects of language which form the subject of this paper follow the same principles. A severe lesion of any part of the brain which gives origin to the physiological processes underlying symbolic thinking and expression may destroy all forms of speech; the patient becomes dumb and cannot read or write. But if the lesion is not associated with widespread disorder of structure or physiological function, some power will certainly return, and the various tests may not be uniformly affected. Finally, when the period of neural shock has passed away, the abnormal manifestations may be confined, for a time at any rate, to disturbance of some one or more aspects of symbolic thinking. These are not the primary elements out of which speech has been evolved, but the forms assumed when the complete and highly developed function is broken up.

The majority of observers have fallen into the error of supposing that these dissociated manifestations revealed the elementary basis of the acts of speaking, reading and writing. On the contrary, they show the components into which the complete psychical procedure can be split up. At first the whole of symbolic thinking and expression may be rendered impossible, but gradually certain forms return, whilst the recovery of others may be indefinitely retarded. When a man has received a severe injury to his foot, at first he may not be able to walk at all; but after a while he is found to be walking in a peculiar manner according to whether the wound affects his toe or his heel. The gait he assumes is not an elementary component of his normal method of walking; it is due to the fact that he cannot place some one part of his foot on the ground.

Provided we bear these principles strictly in mind, we are justified in recognizing the existence of various forms of aphasia. For the clinical manifestations are so obviously different, according to whether the loss falls mainly on one or other aspect of symbolic thinking, that some differentiation is necessary. But it must be clearly understood that the highest functions are first and most severely affected; tests which are less difficult may be carried out perfectly, although they seem to depend on the same act of speaking, reading, or writing.

§ 1.—*Verbal Defects.*

In severe cases of aphasia the power of using words both in external and internal speech is gravely affected. The patient may be reduced to "yes" and "no," together with a few stereotyped phrases or emotional ejaculations. When due to some lesion originating within the substance of the brain, so profound a disorder of speech is usually accompanied by other defects of language. It is the less severe injuries that reveal a loss of the verbal aspect of symbolic expression in an uncomplicated form.

Amongst my patients with gunshot wounds of the head were three who showed this disability alone. They were unable to produce at will and without effort the word required, although they could recognize it when it was offered to them either verbally or in print. If they were able to utter the requisite word, they could use it correctly for naming objects, although it might be so badly pronounced as to be scarcely recognizable. They could repeat just as much, or perhaps a little more than they could say voluntarily, and executed without difficulty commands given by word of mouth or in writing. All the remaining forms of symbolic thinking were carried out perfectly and these patients were acutely conscious whether the words they uttered or otherwise used were rightly or wrongly produced.

Let us examine more in detail the manifestations of this purely verbal form of aphasia. External speech is profoundly affected; one patient, who was admitted to the London Hospital five days after he was wounded, could not utter a single word. He rapidly improved and within a fortnight was making sounds which bore a remote relation to the words he was seeking. Both my other patients appear to have passed through this stage, but they came under my care after they had already gained a considerable vocabulary. But it is these slighter disabilities that throw the most light on the true nature of this affection of speech; they not only allow us to study the various forms assumed by defective verbalization, but also permit us to hear some account of their difficulties from the patients themselves. Fortunately No. 17 and No. 4 were highly intelligent officers, who were able to give valuable introspective information.

No. 17 said, "At first I don't think I had more than a twenty-word vocabulary." "I want to say a word, but it is at the back of my mind, I can't dig it up. I sometimes have to alter a whole sentence because I have difficulty in finding the word." He frequently substituted a more metaphorical expression for the usual and more direct word, as for example "dig it up" for "remember." This tendency can be observed in the speech of many normal Englishmen, who are unaccustomed to choose their words carefully.

No. 4, who from the beginning showed a less severe disturbance of speech, said he found difficulty with "tenical terms" (technical terms). "Yesterday I had diff-ulty in remembering what you do with a skull — — Tri — — Tre — — Trephine." "I'm confine (confined) to the words I've got back."

This condition leads to gross faults in pronunciation; No. 6 spoke of "the claration of war by the Ollies" (the declaration of war by the Allies), and all these patients were liable to lapse into similar errors.

These always assumed the character of defective word formation; the sentence might be halting and words were either lacking or mispronounced, but the essential rhythm, both of word and phrase, was intact.

But it must not be supposed that the loss of function in such cases was purely articulatory. Internal speech was profoundly affected; writing showed exactly the same faults as uttered speech. Thus No. 4 said, "At first I used to talk, missing out words; now I don't do that." As a matter of fact this tendency still existed at the time of this confession and was evident when he wrote to dictation. For he complained, "I can't carry many words at a time. If you give me a phrase containing four words I can do three, and then have to get you to dictate further. I find on reading over I leave these small words out, quite often."

When No. 17 was asked to read a short printed passage silently, and to write what he had learnt from it, he wrote slowly, and with great effort. He then said: "I always have to spell out every word, even the little ones. I have to say 'of'; I know it is a preposition, but then I have to think: is it 'to,' 'from,' or 'of'? Prepositions are always a bother to me." "It's not the long words that stop me. It may be words of four or five letters. It's not specially the long words that are the trouble. When I am puzzled over a word like 'help,' I have difficulty in knowing if the l or p come last. My bankers are Holt and Co.; at first I had difficulty in knowing whether the l or the t came last. Now I've written to them so often, I've learnt it." "My spelling of Latin derivatives is better than that of other words."

"I can't spell. I think I can write if I have tried on the blotting paper. I have difficulty in finding the words, because I speak the thing out when I write it. When I write a letter hurriedly, I hurry on and leave out lots of to's and at's and words. The easiest way for me to correct is to get somebody to read aloud, and I say, 'Wait a minute, I want to put in an 'of' or a 'to.'" "I'm teachable. If I spell a word wrong, my wife tells me; I get it right. When I wrote to my bank man-ger (manager) I always spelt 'man' and 'ger.' She told me that; now I get it right." Here his pronunciation of manager was exactly as he had been in the habit of writing the word. "It's no use my trying to spell out the words when I'm blocked with a word. But if I can visualize it, I'm all right; visualize the word as it is written."

The form assumed by written words corresponds in a remarkable way with the errors of external verbalization. In the speechless stage these patients write with extreme difficulty, and may be reduced to their signature only. As they improve in speech, the power of writing increases, but it tends to show defects analogous to those of pronunciation.

The most striking evidence of the disturbance of internal speech was given by the hand, eye, and ear test (Table VI). These patients found considerable difficulty in imitating my movements correctly when we were face to face. At first sight it might seem as if this had nothing whatever to do with speech. But analysis of our own experience under similar conditions shows that such imitative movements are accompanied by more or less internal verbalization. Although we may not be conscious of saying to ourselves "eye" and "ear," we undoubtedly formulate some such words as "right" and "left"; and this is fully borne out by the patient's own statements.

When, however, he is told to imitate my actions reflected in a mirror, he can carry out the test perfectly without hesitation. For under such conditions the movements can be performed correctly without the use of words. They are direct and intuitive. No verbal symbol intervenes between the reception of command in gesture and its execution.

In order to test the truth of this explanation I asked the patient not to imitate, but to record my movements in writing. This he did badly, sitting face to face as might have been expected from previous experience. But when told to write down my actions, as seen in the mirror, he found exactly the same difficulty. Writing necessitated the interpolation of words into what would have been otherwise an act of non-verbal imitation; this spoilt the response to a test that had otherwise been carried out perfectly. No. 4 explained: "I say to myself, 'left hand'; then I have to say to myself: 'left ear or eye.' When I've said it, when I've decided, I can write it quite easily."

An exactly similar result is obtained in this group of cases, when the order is given pictorially. A series of cards are prepared, each bearing a drawing of the essential factors in one of these movements; these are handed to the patient in order, and he is asked to carry them out without speaking. If he holds the card in his hand, as he would normally look at a picture, the records are bad, just as with face to face imitation. No. 17 said: "It's like translating a foreign language that I know, but not very well. It's like translating from French into English."

But when the figure on the card was reflected in the mirror, his imitative movements were carried out perfectly, and without hesitation.

For no verbal intervention is necessary between the visual reception of the order and its performance.

These patients suffer from a defective power of verbalization; they cannot produce with certainty the word they want either for external or internal speech. They usually appreciate the significance of words presented to them, and can therefore execute both oral and printed commands; but they are liable to become confused in general conversation, or if the order is given too rapidly. No. 4 said: "Some words, when I listen to them, I can't collect the meaning. Nurse said something about your coming this morning; but she said it too quickly, I couldn't collect it; she had to say it again."

An exactly similar difficulty occurs when they attempt to read to themselves. No. 17 said: "I can't read a book to myself because I'm bothered when I say the words. I can get the meaning of a sentence if it is an isolated sentence, but I can't get all the words. I can get the middle of a paragraph. I have to go back and start from the preceding full-stop again."

Reading aloud conveyed the meaning better than when he read to himself; for the sound of the words, though badly pronounced, reinforced his understanding of the significance of the paragraph as a whole.

Writing to dictation is slow, and words may be omitted. No. 4 said: "I can't carry many words at a time. If you give me a phrase containing four words, I can do three, and then have to get you to dictate further—or read it again." On reading what has been written to dictation, such patients notice the omissions, and attempt to correct them. They find no difficulty when the various combinations of the man, cat, and dog test are dictated, and never substitute one word for another, as is so commonly the case in other forms of aphasia.

The verbal aspect of numerals may be affected, but not their significance. Thus, in the early days after the injury the patient may be unable to count, because he has not those verbal symbols which are numbers. With gradual recovery this is replaced by various troubles with compound numbers, and inability to carry a long list of figures in the memory. Thus No. 17 said: "When I first began to look at reference books like the Army List, I couldn't say it was 982 when I looked the page up; but if I didn't try to say it, I could look up the page without difficulty. When I tried to say it I would say different numbers, and then I would muddle myself and have to look it up again. Silent thought was easy, but vocal thought was muddling."

He played an excellent game of bridge, but complained: "When I am scoring in a game, I say 28, when I mean 41. But this makes no difference to my score. I count 41, though I may say 28. I do the sum right, but get the figures I say wrong." Addition and subtraction were correctly carried out, and he kept his own bank-book.

No. 4, however, found some difficulty in carrying over a number when adding or subtracting, although the actual calculations were correctly performed. He had worked in a bank before the War, and was accustomed to add up long columns of figures in his head; this he now found difficult. Such loss of capacity to verbalize figures may lead to actual faults in simple sums if the disturbance is severe and the patient poorly educated [No. 6].

None of these patients failed to name coins correctly if they could find the requisite words; however badly pronounced, the sound bore some relation to the actual name. They could always indicate the relative value of two pieces of money placed in front of them, even if they were obliged to do it in dumb show on their fingers.

Provided they could speak sufficiently, they had no difficulty in naming a series of common objects or even geometrical forms, if sufficiently well educated. They could choose any one of them either to verbal or written command, if the order was given in one word, and not in a complex phrase.

Asked to write the names of such common objects or of colours, without saying them aloud or under the breath, the words produced were recognizable, and corresponded to the object shown; but they displayed the same faults as if they had been pronounced. Thus, pyramid became "pymared," "pymerad"; scissors "sissiors" and ovoid "oboid" throughout one series of written tests.

Drawing is not affected and the outline of a simple model can be reproduced from memory. Told to draw an elephant the result is recognizable and exhibits all the salient parts of the beast. These patients have no difficulty in drawing a plan of some room with which they are familiar; this is a true ground plan and does not show that tendency to lapse into details of elevation so common in other forms of disturbed symbolic thinking. They have no difficulty in finding the way, and can describe a journey from one place to another without difficulty or confusion.

They can look at pictures with pleasure and understanding. They are able to appreciate jokes expressed pictorially or in print, and are capable of playing games at an early stage of recovery. No. 4 found "jig-saw" puzzles very "re — edji — kate — ive" (re-educative).

One of the most striking faculties possessed by this group of patients is the power of recognizing their errors. On one occasion, No. 17 remembered that six months before he had been forced to substitute another word for "economy," which he had read but could not remember. On looking up the actual record it read as follows: "I never found we were much in pocket over her — her — thrift or her — I want another word but I can't get it."

Innumerable examples might be collected of this power of appreciating whether an act of speech is rightly or wrongly performed. It is one of the most characteristic features amongst the manifestations of verbal aphasia.

§ 2.—*Nominal Defects.*

In this disturbance of symbolic thinking and expression, we are not dealing with a difficulty in forming words, but with a disorder of their nominal aspect. A name is a pattern which, if appropriately chosen, fits an external object or state of things around us. When a set of geometrical figures are ranged before me and a cone is placed in my hand out of sight, I know that it resembles one of the objects on the table, and this in spite of a diversity in size, material or colouring. A pattern has been produced as the result of the sensation from my hand, which agrees essentially with what I see. If, however, I give the object a name and call it a cone, I evoke a symbol which indicates that pattern. In the same way, when I give the time as five minutes to eight, I not only utter a series of words, but name a position on the clock; in a few minutes the long hand will reach the hour, and this position is consequently named "five minutes to eight." It differs fundamentally in nominal significance from "five minutes past eight"; the two are as diverse as a cone and a pyramid appreciated by touch.

During the first few weeks, the patient may be almost dumb, with little or no spontaneous speech and unable to write even his signature. But with the passing away of the effects of neural shock he regains his power of speaking. Should he be suffering from a disorder of the nominal aspect of symbolic thought in an uncomplicated form, his enunciation will become almost normal, except where he has difficulty in naming. There is not that profound disturbance of pronunciation which runs through all the utterances of the verbal aphasic.

Thus, exactly two and half months after his wound, No. 7 was unable to name a single one of the common objects presented to him in order; he moved his lips like a person seeking for a word and said: "I can't say it. I think I know it, but I can't say." In so far as he could find words to express himself they were correctly pronounced. He was unable to repeat these names, although the sound he uttered bore a distinct relation to the word said by me; in some cases it came so close that it might be considered correct. For example, key became "kay," pencil "pintil," matches "mats," and penny was twice repeated accurately. Asked to repeat "scissors" he says "sis—I can't open far enough," pointing to the jaw.

He could not write the names of any of these objects, but he could copy them from a printed card in capitals only; he could not translate the words into cursive handwriting, for this seems to demand a power of consecutive nomination.

When I named one of the objects on the table in front of him, he chose slowly and was evidently puzzled; in three instances he gave up altogether, but in nine out of the twelve observations he made a correct choice. On presenting him with the same words singly in print he failed to select any object on six occasions, but chose correctly, though with hesitation, in the remainder.

When however he was shown one of these common objects or when it was placed in his hand out of sight, he never hesitated for a moment; in every instance his choice was correct and prompt.

This disability does not depend on destruction of images but on a want of names. No. 2, who had reached a further stage of recovery, could choose correctly to verbal command, although his power of naming was very defective. So perfect was his memory of the order in which the objects stood upon the table, that with his eyes closed he could point to each one in turn as I named it; but asked to give their order in words, he was completely unable to do so.

When this patient was told to say the days of the week, the first four were correctly given, but Friday was called "Fysday" and Saturday, "Second—Septen-day—Sutton-day": then after a pause he remarked "Sunday is the only one I can remember after that." Finally, after three attempts he went through the week correctly except that "Second-day" replaced Saturday. He was then given a series of cards, each of which contained the name of one day in print; these he copied perfectly in capitals, letter by letter. His lips did not move and this was obviously pure imitation. When, however, he attempted to translate these printed words into cursive handwriting, he muttered each word under his breath and wrote, "Mondey, Tuersty, Wednersty, Thersteay, Freray, Satereay, SunDay." Asked to read the printed words aloud he did so with remarkable correctness, until he came to Friday, which was first called "Fys-day" and then "Friday"; he said "Secon-day" but changed it to "Saturday" and finished with "Sunday" perfectly pronounced. When he attempted to write the names of the days of the week to verbal dictation, he failed badly and most of them were left unfinished ; Friday, however, he wrote correctly. Told to write them spontaneously, he was equally unsuccessful, although each word began with the appropriate letter. He was then told to repeat the days of the week after me, and to write each one as he said it. His enunciation was perfect, and although he continued to say the word correctly all the time he was attempting to write, the results were little if at all better than when he wrote spontaneously.

Here we see that correct verbal imitation was possible either when repeating the sound of the words or when copying print: but any act which required the intervention of the nominal aspect of symbolic thinking or expression was badly executed. Worst of all was writing to command; for this demanded not only the power of naming, but also the translation of the name into written symbols.

Except in the earlier stages, these patients can read to themselves, but with great slowness and uncertainty. They are particularly liable to grave misunderstandings due to defective appreciation of the nominal value of the words they are reading.

If what they are given to read silently is in the form of a command, this want of appreciation becomes still more apparent; for not only must the patient understand the printed matter, but he is compelled to formulate the meaning to himself before he can carry out the order that lies behind it.

No. 7 was given a series of cards, one by one, each of which contained a simple order such as "Shut your eyes," "Touch your nose," &c. He was told to read it to himself and to "do what it says." This presented great difficulties ; sometimes when told to shut his eyes, he brought his hand to his eye, or asked to give his hand, he touched the left with the right. Once only in eight attempts did he carry out such orders correctly. Reading them aloud seemed if anything to cause greater difficulty, and copying the legend on the card did not materially improve his power of carrying on the action demanded. But, as soon as the commands were given him verbally, he made one partial mistake only, touching his eyes with his hand, instead of closing them.

Reading-aloud seems to add to the difficulty and is no aid to understanding, as with some other forms of aphasia. The patient tries to spell out the words and becomes confused because he is uncertain of the nominal value of the different letters. At a time when No. 2 could both say the alphabet correctly and made no mistakes in writing or reading it, he was unable to read aloud the man, cat and dog series. Given "The man and the dog" in print on a card, he said, "T-H-E.: the next one is M-A day, no D. This is A. This is N. This is — — D T and H and E. This is D. That is O and T. I'm not sure about that one." Asked "What does the whole say?" he answered "The only thing I can tell you is it was a dog." This is a fair sample of a series of observations on his power of reading aloud a combination of simple words not containing a command.

But when pictures of the man, the cat and the dog were shown to him in series two at a time, he never hesitated for a moment but named them accurately. Evidently he still possessed the power of evoking these simple words as symbolic representations of pictures. But when offered the names in print he was confused by want of certain appreciation of the value of each letter. He had no difficulty with the alphabet as a sequence; it was the nominal value of any one of its constituents which was uncertain.

In these observations all the words were monosyllabic and recalled a child's first reading book; much of the difficulty arose from defective comprehension of single letters. The same objects represented pictorially could be translated with ease into their nominal equivalents. But as soon as pictures were made to carry a command, as in the hand, eye and ear test, the action demanded is as badly carried out as if the order had been given in print. The necessity for formulating the significance of the picture gave it a high nominal value.

The nature of this disturbance of symbolic thinking comes out particularly clearly with the clock-tests. There was not the slightest difficulty in setting one clock in direct imitation of the other; even the short hand was moved into a proportionate position between the two numbers of the hours. But none of these patients could tell the time or write it correctly. They set the clock badly to oral command or when the order was given in printed words; but when it took the form of figures, so called "railway time," it was perfectly carried out. For to set a clock when given such a number as 4.45, the patient first places the hour hand at 4, and then sweeps round the other one to the position he associates with 45 minutes; but if he is asked to set "a quarter to five," the temptation is to set the one hand at 5, whilst the other hovers uncertainly between "a quarter past," and "a quarter to." This difference does not, however, apply when the patient attempts to tell the time; it is equally difficult for him to evoke the name "4.45" as "a quarter to five."

Throughout these tests there is a profound tendency to muddle up "to" and "past." The number of the hour has an overwhelming attraction; for example, when setting twenty minutes to six the long hand may be rightly placed, but the hour hand points to six. Not infrequently the nominal value of the two hands was mistaken and the long one set to the hour.

All this does not signify that the patient does not know the time. No. 2 said, "That is, when you eat," or "when we were there," of some particular event, and is always right, although he cannot reproduce the time in either case. Moreover, when shown the test-clock set at some definite hour, he could pick out a card on which the corresponding time was printed in figures, ten times in succession without a moment's hesitation. It is not knowledge of the time that is lacking, but the symbolic means of expressing even to themselves what they know; at the same time they have difficulty in understanding the nominal aspect of words, spoken or printed, in which they are told how to set the clock.

Although it is impossible to name an object with any certainty, the patient may be able to do so by using some metaphorical expression instead of a single word. For example, during the tests with colours, No. 2 was wrong in eleven out of sixteen attempts to name one of them set before him (Table II, p. 97). So bad were his mistakes that he might have been thought to be colour blind; for white was called "green," black "red," and green "blue." Exactly the same kind of error occurred when he chose a colour from its printed name on a card; in this case he even chose white for black, and black for green. When asked to read them aloud eleven out of sixteen were wrongly named.

From the observations we might be tempted to think that he was devoid of all knowledge of the nature of colour, in fact that he was "mind-blind." But in his attempts to explain to me his difficulty in reading the printed cards he began to point to my white coat, his khaki tie, the blue band on his arm, which he wore as a wounded officer, and the green of the trees just outside his window. Instead of the names of the colours, he was encouraged to use a series of similitudes; black was "what you do for the dead," red "what the staff wear," or, pointing to the lapel of his tunic, "where the staff have it"; blue was "my arm," green "what is out there" (pointing to the trees), white is "what you wear" (a white coat), yellow "this one" (holding his tie). Twelve times he was shown cards bearing the name of one of these colours, and in every case he gave the right answer.

I then reversed the procedure, saying to him "the dead" and he chose a black piece of silk from a set of colours on the table in front of him; for "what is on your arm" he selected blue, "what the staff wear" red, "what is out there" green, "like your tie," which he was not allowed to look at, led to his choosing yellow. For violet, we had agreed on a lamp-shade that stood by his bed; this had been removed from the room before these observations began, but to the words, "like a lamp shade," he chose violet. Throughout he made no mistakes in fourteen consecutive tests. This shows that he had an acute sense of colour, but that names were wanting for expression, and were badly understood when presented, both verbally and in print.

The power of counting or uttering numbers in sequence usually returned step by step with that of saying the alphabet. No. 2 had no difficulty with either when first he came under my care; but this did not mean that he could employ any particular numeral directly and with certainty in arithmetical operations. He could pick up five things when told to do so, but asked to add five to any number, he would count on his fingers, or would place five dots on the paper; the figures 2, 3, and 5 rapidly became associated with two, three and five dots, which he visualized around them; but 4, 6, 7 and 8 were counted on his fingers or dotted on the paper.

He confessed that numbers puzzled him at first; if "eighteen" was said, the meaning did not come until he had counted it up. In the same way, he could say the alphabet perfectly, but the nominal value of any single letter was frequently defective. This difference in the facility with which a name is appreciated in series and as an isolated word is familiar to all of us in a foreign language; many who can rattle off French numerals without difficulty, experience a moment's hesitation when told that the price is "quatre vingt douze francs."

No. 2 experienced the same sort of difficulty when he was informed that some engagement was fixed for "Tuesday the twentieth of March." Telling him the day of the week or the month did not convey to him the part of the week or of the year in which it fell.

All three patients failed to carry out even simple arithmetical exercises. They could add 356 to 231 on paper, but all of them failed over such a sum as adding 275 to 856, which required the carrying over of a number from one column to the next. Captain C. (No. 2), who was by far the most highly educated, counted up to five, and then on for another six, giving the answer correctly as eleven ; he then counted seven, and one more, making eight; from this he went on for five more, saying "thirteen." He wrote "3" but was wrong with the last column of figures because he forgot to add the 1. Both the other patients wrote down the sum of each pair of figures separately ; thus No. 7 wrote :—

$$\begin{array}{r} 2\ 2\ 8 \\ 7\ 3\ 4 \\ \hline 9.\ 5.12 \end{array} \quad \text{and} \quad \begin{array}{r} 3\ 4\ 5 \\ 8\ 6\ 8 \\ \hline 11.10.12 \end{array}$$

All of them broke down completely when asked to do simple subtraction, and No. 7 took the lowest number from the highest, whether it was in the upper or lower line of the sum ; No. 18 always worked from left to right throughout.

As might have been expected, there was considerable difficulty in finding the usual names for coins, although it was obvious that they were still recognized correctly. Thus, a sixpence was called "half a shilling," two and six was "two and a half," a ten shilling note was "half a pound" ; and yet, in a series of twenty-four observations, many coins were given their usual names, and there was no evidence that any one of them was mistaken for another of a different value.

But when the attempt was made to give the relative value of two coins, such as a sixpence and a shilling, laid in front of him, the patient became confused; he tended to add them, saying "one and a half." With 1d. and 6d. Captain C. said "seven." When, however, the question was put in the form, "How many of that [6d.] would you have to give me for that [1s.] ?" he answered "Two: one more of that [pointing to the 6d.] to get one of that [the 1s.]." Then he suddenly burst out, "One more sixpence to get a shilling." Throughout this series of tests he could never name the relative value directly of any two pieces of money, and yet by these indirect means it was possible to show that he understood perfectly. No. 7 showed exactly the same difficulty in formulating the relationship, but was able to pile up a heap of coins on the one side which exactly equalled the larger one in value. Given a shilling, he collected together a sixpence, a threepenny bit, and three coppers; a two-shilling piece was placed before him, and he chose from the money on the table a sixpence, a threepenny bit, a shilling, and quickly added three coppers. Yet when asked, "How many of this [6d.] go into that [2s.] ?" he said, "Two — two — two shillun —," and with 1s. and 2s. replied, "Three shillun." But behind this difficulty in expression lay a correct and intuitive comprehension of the relative value of the coins to one another.

Nearly two years after the injury Captain C. (No. 2) was able to fill up a cheque spontaneously; but he could not be certain that the written words and the figures corresponded with one another. On the occasion when he drew a cheque in my presence for eighty-five pounds, ten shillings and sixpence, he filled in the figures as £80 10s. 6d. He noticed this discrepancy and succeeded finally in making the correction; but the cheque would not have passed the bank.

All these patients could draw from a model, and, after its removal, reproduced the drawing from memory. But when No. 2 and No. 7 were asked to draw an elephant, the result bore no relation to this animal; it was without trunk, tusks or ears. This was the more remarkable as Captain C. (No. 2) used to amuse himself by drawing pictures of the animals he had shot in Cashmir; on one occasion, when talking about transport in the East, he drew spontaneously a spirited rough picture of a camel. Visual images are not destroyed, but they cannot be evoked with certainty at will or to command.

This is particularly evident when the patient is asked to draw a rough plan of some room he knows well. No. 7 was asked to put down on paper the relative position of the objects in the ward he could see from his bed; he failed completely. But when I drew an oblong on the paper, saying, "That is your bed," and asked him to point to the situation of the various things he could remember, he was astonishingly accurate. For instance, he pointed to the left, saying, "There's only my bed," and to the right, "There's a bed; no one there now"; he then indicated the head of the bed, and touched the wall of the examining room in which he was at the time, to signify that here was a wall. All these details and many others were correct, but he could not put them down himself on paper. Captain C. (No. 2) started to draw a plan, but filled it in with details in elevation. Images were certainly intact, but they could not be reduced to a symbolic formula.

Orientation is unaffected and such patients have no difficulty in finding their way, provided they are not asked how they intend to go from one place to another. No. 2 succeeded in explaining this to me as follows: "I have no streets in London; no names at all. Suppose I was going from the Army and Navy, that's what it's called, just round here (the Army and Navy Stores), I should then say some place to a hospital about a quarter of a mile away. I remember the hospital is on the left on the way to the War Office, about half-way. I believe it's the Abbey. No, it's near the Abbey on the way to the War Office." Later he added, "I saw the Hospital." I put as a question, "The Abbey?" and he replied as follows: "It's here, but then it's gone again and I have to feel for it again. The only thing I can remember of it is the opening, the big opening, where everybody goes in. I can get that" (moving his hand in the form of a large arch). Since it was obvious that he had described Westminster Hospital and Westminster Abbey, I asked, "Have the Hospital and Abbey anything to do with one another?" He answered, "Nothing except my focus, the place of them; the distance, that is all. I should say how far the Hospital is to the Abbey in re- —, in re-, — that's where I go wrong. I want to say in re- something [relation]. There are just little bits in expressing what I want to say; little bits in which I have to turn my brain another way to get what I want to say, whereas a year or two ago I should have said it without thinking. You see; it's like this: with me it's all in bits. I have to jump like this," marking a thick line between two points with his pencil. "Like a man who jumps from one thing to the next. I can see them but I can't express. Really it is that I haven't enough names. I've got practically no names. The easiest thing is what I do now. I say what I can, it's all wrong; but they get an idea of what I want to say."

It is evident from this conversation that he was able to recall visual images of objects on his way from the Empire Hospital in Westminster to the War Office in Whitehall. First of all, he saw the Army and Navy Stores and then Westminster Hospital on his left, with the great door of Westminster Abbey on the right; but want of names prevented him from connecting the two, except in position, and he was forced to jump from one image to another without the cohesive links of verbal formulation.

Pictures apparently conveyed their full meaning so long as they did not imply a command to carry out some action. No. 18, when shown a picture of an aeroplane looping the loop, explained the manœuvre by pointing and gestures, helped out by words. No. 2 saw all the points in a Bairnsfather picture of "Old Bill" boxing with Hindenburg. But jokes frequently present considerable difficulty if they demand nominal formulæ for their comprehension.

These patients enjoyed games and could play dominoes and draughts. Captain C. (No. 2) was above the normal average at chess, but could no longer play bridge. "The names of the cards bother me," he said. "It's just names; I used to play a good game of bridge."

§ 3.—*Syntactical Defects.*

This form of disturbance of language can be distinguished by the fact that the patient talks jargon. In either verbal or nominal aphasia he cannot easily evoke the words and names he desires to use, and consequently speaks slowly and with difficulty. In this group, on the other hand, he talks with great rapidity when once started; the words may be recognizable but badly put together, or, if the loss is more severe, they may be pure jargon. The rhythmic movements of the phrase are affected; they are hurried and slurred, and the patient cannot "touch off" the words so as to produce a correctly coherent sentence. In the lighter forms of this defect there is no difficulty in finding words or names, which may be intelligible, especially if the subject of the conversation is known. Thus, when No. 15 was given quinine as one of the tests for the sense of taste, he said, "Rotten to drink it. Something medicine or that. Make you drop of water after it, so to take out of your mouth." Sometimes speech closely resembles baby language; for example, asked what his right hand felt like, No. 13 said, "Tiff-rent from uffer 'n — ka tell ooh, know zis un seems strong."

But at times even this patient became unintelligible; describing to me a picture of a tramway car with a woman at the wheel and a man conductor he said: "Here's lay, here handle, the man conduker, on the nines, shot steats on it, zee passengers, two man, lady" (Here is the lady; she's at the handle; the man conductor. On the lines; it's got seats on it. Three passengers, two men and a lady.) He then read the the legend at the top of this picture as "Mıns bixet o-er men" (Woman's victory over man).

The power of naming an object in sounds that are comprehensible varies with the severity of the affection. Thus, in No. 13, single words could be produced perfectly as names so long as they were applied to familiar objects; but when attempting to name colours he "jargoned" orange, green and violet; in every instance, however, the sound was recognizable as a name. No. 15 was extremely unstable in his nomenclature, using "blacking," "pencil" and "black-lead" indiscriminately for pencil. In naming colours he also found difficulty with orange, violet ("volley") and to a slighter extent with green; but all the truly monosyllabic colours he named correctly.

But in No. 14 the sounds, though emitted volubly, seemed to bear no relation to the name of the object; thus, matches became "Stes-missness." He then seized a pencil and wrote "match," showing that he knew the name. With a penny he shook his head, said "No" but wrote the name correctly; in some instances, however, he could neither say it intelligibly nor write it at all. Here the jargoning had become so severe as to destroy all power of producing some names at will.

He could say "yes" and "no" correctly, and answered "Come in" to a knock at the door. He was extremely intelligent in the manner in which he employed his jargon, and was particularly fond of using "there" and then pointing to the map, the book or the picture to which he wished to refer. He used words or short phrases, but could not amplify or repeat them at will. On September 15, 1915, he was reading a French account of Napoleon's Russian campaign, and wanted to tell me how closely the dates coincided with the Russian retreat which was then taking place. He took the book in his hand, pointed to the date "15 Septembre," saying, "Now — just the same — there — yes — over there."

One day during a set of observations he said, "Funny thing — this worse — that sort of thing." Then he seized his note-book and wrote, "as, at"; I asked "You mean conjunctions and prepositions?" and he replied, "Yes — that sort of thing."

The grammatical structure of the phrase is liable to be badly affected. The patient talks fluently in short jerky sentences, slurring or omitting many of the junction words. Even when they are present, it is difficult to hear the articles, prepositions and other components of syntax. Asked what he had done since his admission into the London Hospital, No. 15 said, "To here—only washing—cups and plates. That's about all you've got to do here." "Have you played no games?" "Played games—yes—played one, daytime, garden."

The power of writing suffers on the whole less than external speech, because the defect is essentially one of balance or rhythmic utterance. Thus No. 14 made unintelligible sounds when asked to name a set of common objects lying on the table before him; but in thirteen out of eighteen serial attempts he succeeded in writing names for them, which were recognizably correct. Single words used as names were so much more easily written than spoken, that this patient always carried about paper and a pencil to help him in his conversational difficulties.

After the acute phase of shock has passed away, it is always possible to obtain some recognizable signature, and usually the address can be correctly written. But No. 15 who wrote his name and address perfectly could not write that of his mother, in whose house he lived; the unfamiliar beginning inhibited the almost automatic remainder of his action.

Patients with a severe degree of this disorder cannot write a letter, but in the milder cases such spontaneous writing is carried out far better than might have been expected from the character of external speech. This is evident when the patient is asked to read what he has written; No. 13 wrote a perfectly coherent and well-spelt letter, but jargoned badly on attempting to read it aloud.

The power to write from dictation shows all grades of disturbance from almost complete loss in No 14 to a difficulty with consecutive phrases, but not with isolated words or sentences. Thus No. 13 succeeded perfectly in writing the man, cat and dog series to dictation, but failed badly with a passage chosen by himself from the newspaper.

All these patients could copy correctly and transcribed print with cursive handwriting without hesitation.

Reading to themselves was their favourite recreation and, although they showed evidence of remarkable powers of comprehension, they were hampered by the disorderly structure of the phrase. No. 15 confessed, "Don't always know what they mean going to talk to." No. 14 followed all the movements at the Front on large scale military maps, and enjoyed demonstrating to me the changes reported day by day in the newspapers. But any attempt to impart this information verbally revealed his disabilities in an acute form. Moreover, he was quite unable to follow up a conversation suggested by what he had read to himself and understood. After he had pointed to "15 Septembre" in his French book on Napoleon's Russian campaign, I answered, "Yes, that was also a beautiful summer and the snow came early." He replied, "Yes—oh! did it?—oh! yes." He showed so much interest that I asked him when Napoleon first had difficulty with the snow; but he turned the pages aimlessly. I then questioned him as to the date on which Napoleon reached Moscow (September 14, 1812), the starting point of our conversation, but he shook his head and could not answer. Spontaneous thought was rapid and correct, but the power of reproduction was evidently defective.

The following incident in his life whilst under our care shows how accurate may be the processes of intuitive comprehension even with a severe syntactical disturbance. He was walking in Kew Gardens with the medical officer in charge of the Hospital, when he pointed out a new variety of heath. The doctor said, "Scotch," to which the patient answered, "No, no, you and me." It was an Irish heath and both the patient and his companion were Irishmen.

The nature of this disturbance of symbolic thinking was clearly demonstrated by a series of observations carried out with No. 13. He chose a picture from the daily paper showing a box for the receipt of flowers for wounded soldiers, placed on the platform of Snaresbrook Station. Underneath was the legend, "This box has been placed on the platform at Snaresbrook Station as a receptacle for flowers for the wounded. Many of the passengers contribute nosegays daily from their gardens, and these are forwarded without delay to the Bethnal Green Infirmary." As a Londoner in an East End hospital this interested him greatly.

Asked to write down what he had gathered from his reading and from the picture, he wrote, "At Sanbrook station they have large box which are collicting flowers for the wounded soldiers, and they are sending to the Velnah Green Hospital." I dictated the printed description with the following result: "This box has been placed on the platform a smatbrook station as sesful for the wounded many of the passengers contic nonsgay from there garndens and these without delay to the Belnelth Green imfirmary"; but he was able to copy the printed sentences in perfect handwriting without a fault or omission. Given the paper and asked to read it aloud, he produced the following jargon: "Zis box had been place on the plakform at Senbrook Station as a . . . for flowers for the wounded . . . Many ob le pasn-gers contibute nosezays from their gardens and these are for-boarded without delay to Besnal Green Internary. . ." Shown the picture, he pointed out all the various significant objects without hesitation, giving them recognizable names. "Box, Flowers for wounded shoulders. On the back, plakform, Senfbrook. Ladies see, putting flowers into box."

He evidently understood the significance both of the printed legend and of the picture, but he could not reproduce his conception in coherent words or in writing, although he could copy correctly.

In the more extreme cases, disorders of expression make it impossible to test the power of reading or writing from pictures. With a less severe disturbance, however, a simple test, such as the man, cat and dog series, can be carried out, when two of the three pictures are presented to the patient together. But the response, both oral and in writing, tends to take the form of the correct names with no junction words. Thus, No. 13 had been through the variants of this test in which he had read aloud and had written to dictation a series of combinations of the man, the dog and the cat. But when shown simultaneously any two pictures, for example, the man and the cat, he replied systematically, "Man, Cat," &c. After this series, expressed in spoken words, he was again shown the pictures and asked to write silently what he had seen; he wrote "Man, Cat," without articles or conjunctions.

Another peculiarity of this set of answers was as follows: The normal man when shown two such pictures, that of the man to the left and the cat to the right, frames his answer as if he was reading, and says, "The man and the cat." But No. 13 showed a remarkable inconstancy, both when giving his reply in words or writing. Sometimes he placed the left hand figure first, sometimes that to the right. This inconstance of direction appears again in some of his answers to the clock test given in detail later.

These patients can count and carry out simple addition and subtraction perfectly. No. 14 could make out a cheque for the correct amount spontaneously; but when told to fill it in for a certain sum he inserted figures in the place of the words and wrote, "Five pound 10/6" where he should have written numbers only.

All these patients could recognize the relation between two coins placed on the table in front of them, and none of them experienced any difficulty with change.

The clock tests proved unusually interesting, because, according to their results, the three patients could be arranged in conformity with the known severity of the disturbance. In every instance the clock was set correctly both in imitation and to printed command; to oral command No. 14 made gross errors, No. 15 was slow, but No. 13 made no mistakes. On attempting to tell the time No. 14 failed in every attempt; No. 15 made two errors in substance, but adopted an extraordinary nomenclature; No. 13 was slow but substantially correct. Telling them to write down the time set on the test-clock led to identical results, and No. 15 showed the same extraordinary method of recording the time as when telling it aloud. At one time he wrote 45 to 9, at another 20 past 11; he called 8.10 "2 past 8," because the long hand, pointing to ten minutes past the hour, stood over the figure 2. A similar instability of nomenclature was shown throughout this case, whether he was giving names to common objects and colours or telling the time in speech or writing.

Drawing from a model, or from memory of the same object shortly after it had been removed from sight, was not affected; in fact No. 15 produced a remarkably successful picture of a spirit lamp and said, "I was a good drawder — drawer — at school." But when he was asked to draw an elephant, which he had seen during his service in India, the outline of the body somewhat resembled this animal, but was without trunk or tusks. He proceeded to fill in the mouth, saying the word aloud; then he said "Highons," "Irons" (horns) and immediately added horns to the drawing. After he had finished I asked, "What has an elephant got in front?" He answered, "They carry big trees — — tied round a bit of an iron thing." I then said, "Behind you have given him a tail, what has he in front?" He replied, "He has a big one, quite straight, about a yard long." "What is it called?" "Same what you drink water with." "Has your elephant got a trunk?" "He's lost it," and at once he wanted to add the trunk.

I then pointed to the horns he had drawn on the head of his figure, asking, "Has an elephant got horns?" He replied, "Yes, silver, what you stick out" (pointing to the corner of his mouth and placing his pencil into the position of a tusk). "What are they made of?" "Kind a white bone one, what grows in the mouth — — on the roof — — on the edge of the mouth." Evidently he was confused by saying the jargon words representing horns, and impulsively added them to the figure; but he was finally able to explain his error by gestures giving a correct interpretation of his intention.

No. 13 was more successful, but added the trunk afterwards, saying "Tump."

None of these patients had any difficulty in finding their way, or in pointing out from memory the position of the principal objects in the ward.

They enjoyed games, and both the privates played dominoes well.

They seemed to recognize that the words they uttered were inadequate to express their meaning. Thus, when No. 15 talked jargon so badly that I could not understand him, he drooped his head, blushed and laughing sheepishly did not care to try again to express that particular thought. He was intelligent and useful in the ward, waiting on the other patients, washing up and laying the table correctly for meals; but as soon as he attempted to formulate and express some want he became confused and could not make himself understood. No 14 helped himself out of his conversational difficulties by writing single words in a notebook which he carried about with him. Any gross failure to make himself understood produced an outburst of intense irritability; this was calmed immediately by giving him some test he could perform with ease.

This patient was musical and played me Chopin No. 20 Largo very slowly, reading the notes and giving the change of key correctly. The right hand was clumsy owing to the cerebral injury; but he succeeded in bringing his fingers on to the right notes of the chord, and if he was wrong immediately corrected his error. Keeping his eyes on the music, he recognized by ear when he had struck a false note and that it did not correspond with the text of the music. He played to me other pieces of Chopin, correcting the faults due to the clumsiness of his right hand; but the slow pace of the Largo was in his favour, whilst the complexities of the change in key showed how clearly musical notation conveyed to him the notes intended.

§ 4.—*Semantic*[1] *Defects.*

I have described the various changes in speaking, reading, and writing, produced by affections of the verbal, nominal, and syntactical aspects of symbolic thinking and expression. One other form of disorder emerges from analysis of the clinical phenomena due to unilateral lesions of the brain. This may be called "semantic," because it is comprehension of the significance of words and phrases, as a whole, which is primarily affected.

This has usually been spoken of as an "amnesia," and the patient is said to have lost "the memory of words." But there is no such thing as a "faculty of memory" apart from things remembered; a man who has lost his "memory for words" from a local and unilateral lesion of the brain is suffering from an affection of speech. This may be of the same order as the disturbances of word formation we have considered under the terms verbal, nominal, and syntactical aphasia. The difference lies in the fact that in this case the defect is manifested by an inability to appreciate and retain the full significance of words and phrases.

These patients suffer from no difficulty in pronunciation; intonation and syntax are perfect. They can name common objects correctly, and indicate the one that has been mentioned orally or in print. But when similar experiments were carried out with colours, the answers were less certain, and the patient showed some hesitation and confusion. Oral commands might be badly executed, although the choice made in answer to the printed name was correct. Moreover, he did not seem to remember the relative position of the coloured materials on the table before him; when shown a colour on a printed card bearing the name, he passed his hand backwards and forwards over the row of specimen colours before him until he found the one he recognized. Normal persons rapidly learn the order in which the eight colours lie on the table, and, after the first few experiments, go straight to the one which matches the pattern without groping. This also occurs both with verbal and nominal aphasia; in the latter condition I was able to show that the patient retained a perfect memory of the order in which the colours lay, although he could not name any of them with certainty.

---

[1] From σημαίνειν, to signify. This term was used by Bréal in his well-known "Essai de Sémantique," a study in the science of the ultimate meaning of words. Since I am dealing here mainly with a loss of power to comprehend the full significance of words and phrases together with a want of capacity to use such modes of expression, I have not hesitated to adopt this term, which has already become part of the English language.

The nature of this disorder of symbolic thinking is clearly revealed by the clock-tests. In none of the other forms was there the least difficulty in setting the hands of one clock in strict conformity with those of another; but all the patients of this group carried out this manœuvre slowly, with hesitation, and one of them showed definite evidence of confusion. They could tell the time correctly, but when asked to set the clock in response to either oral or printed command, the errors were extremely gross. The long hand was set as if it was the short one; "to" and "past" were mistaken for one another, and even the hour was wrongly indicated in some instances.

Since the patient could tell the time correctly, his inability to set the clock must have been due to some want of apprehension of the significance of a spoken or written order. No. 10 confessed: "If I can't get the hands exactly where I want, I lose grasp; I get thinking"; and No. 8 said: "I can't make out the difference between *past* and *to* six. I don't know from which side to approach it."

Here we find expressed that inability to recognize significance or to appreciate intention, which is at the root of these semantic disorders. It is not surprising, therefore, that the hand, eye, and ear test was extremely badly executed, necessitating as it does accurate choice between three pairs of possible actions. Moreover, most patients with other disorders of symbolic thinking recognized, when the observer touched his left ear with his right forefinger, that such a movement implied crossing of the hand to the opposite side of the face. One of the most characteristic errors of the semantic group was due to want of appreciation that the action was crossed. Eye and ear might be mistaken, and the patient was particularly liable to give up before completing the action. No. 10 said: "My initial difficulty seems to be right and left; it confuses me; I forget the rest of what I've got to do." Even in the selection of the right or left hand he was obliged to employ a "memoria technica"; "I clench my fist, and then I think of boxing, delivering a blow with the right or left hand; but then, in touching the eye, that feeling is gone."

In all normal persons and with other disorders of symbolic thinking, movements reflected in a mirror can be imitated with much greater ease than when the patient and observer sit opposite to one another. This also applies to the same actions, when given in the form of a pictorial command. But in the semantic group, though the number of errors may be less when the required movement is reflected, there is not the conviction, so characteristic of most of the other disorders of symbolic thinking, that the action is extremely easy. No. 8 said, "When I imitated you I had to think: is that his right hand or his left? When I'm looking in the glass it is easier because the sides are the same; but then I begin to think it out and get puzzled." After he had made an extremely bad record to pictorial commands reflected in the glass, he explained, "Somehow or another I didn't seem able to get the right part of the picture; sometimes I seem to look at one, sometimes at the other. I have to reason out the meaning of the whole picture."

Everything tends to be appreciated in detail, but the general significance is lacking. This is evident when the patient is given a picture, and told to say what he sees in it. He looks at it like a child, pointing out one thing after another, and not uncommonly misses some important feature; asked what it means, he may be entirely at a loss and may then invent some preposterous explanation. No. 5, who was a gardener from the country, chose from the paper the picture of a man riding a cow, over which stood the legend, "Mayor's Curious Steed." He said, "That's a man riding on a colt. No, sir, it isn't, sir, it looks more like a cow — — or a young cow — — no, it isn't — — heifer, sir." After he had read the legend, I asked, "Who is the man?" and he replied, "A farmer, sir. No, Major — — No, the Mayor curse sted — — the Mayor curious stid. It's something you don't see every day, that stid; something very uncommon that animal. It's in a horse's place instead of where it is. I should think myself they are going to show that animal; it's uncommon, that stid, more so to see a man riding on it— —. You don't often see a man riding on a stid." When I asked, "What is a stid?" he replied, "It's something the same family as a cow."

Lieutenant M. (No. 10), an able officer, had been an accountant before joining the Army. I showed him a picture which alluded to the putting forward of the clocks in consequence of the Daylight Saving Act. Standing beside a clock, whose hands point to two, the bride of a day says to her bridegroom, "Look, it's 2 o'clock, and yesterday at this time it was 1," and he replied, "Yes, darling; and yesterday at this time we were two and now we are one!" The patient remarked, "I've got the drift of that. It's not much of a joke. They've stupidly been married; the bridegroom has something stupid to say about yesterday, we were one and now we are two, the same old stupid thing about yesterday, we were two and now we are one — — I could have told the time there better than when you were trying me — —2 o'clock." Q. "What happened at two o'clock just a fortnight ago?" A. "Oh! I see, Daylight Saving Act." Q. "What happened to the clocks?" A. "They were put back an hour. That's a thing I'm rather hazy about. I have to think of that — —forward — — back — —. No, I have to give that up; I've been trying to think that out, and I haven't got to a conclusion yet somehow."

In consequence of this difficulty in appreciating either the full significance of pictures or of printed matter, most jokes become incomprehensible. Suppose, however, the humour consists solely in the detail of the drawing, it is appreciated; but any demand for co-ordination between its various parts or with the text beneath it, meets with little or no response.

These patients cannot play games such as chess, draughts or cards. Nor can they put together "jig-saw" puzzles; No. 8 complained, "I can see the bits, but I cannot see any relation between the bits; I could not get the general idea." This is revealed in another interesting way; he was unable to play billiards because he had no idea how to play off the cushion. He could hit the ball directly, but could not make an indirect cannon.

This want of ability to relate things to one another came out when testing memory for the position of objects in the room or on the table. The patient had no difficulty with his eyes closed in pointing to the position of the window, fireplace, washhandstand, chest of drawers, door, and other pieces of furniture. But asked to say how the washhandstand stood in relation to the fireplace, or the latter to the door, he entirely failed to do so; allowed, however, to say, "The fire is there and the door there," he pointed with complete accuracy. He knows exactly where they are; he is certain that he can "see them" in his "mind," but he cannot express their relative position.

We can only understand the peculiar affection of writing and reading produced by this disorder of symbolic thinking if we bear in mind that the fault is essentially a want of appreciation of relative significance and intention. At first, as in the case of No. 10, the patient may find it impossible to write the name of an object, although he had no other difficulty in nomenclature; but even at this stage he not uncommonly succeeded in writing the first letter of the word he was seeking. At this time he showed a tendency to use the same symbol "𝒬" for the letters D, F, O, R, T and V. Five months later each name was correctly given, but he wrote at a tremendous pace as if in a desperate hurry. The words were carelessly written; cylinder became "cilande" and "cylander," ovoid "ovoad," and cube was shortened to "cub." This excessive rapidity was also evident during the man, cat and dog tests, when he wrote from dictation or from pictures. He said, "I write very hurriedly so as to keep track of it." But if he was given the printed card and asked to copy it, he wrote with much greater deliberation and his handwriting improved considerably.

One form assumed by the errors that occur in this group of tests is the tendency to substitute one word for another; "man" is written for dog, "cat" for man, &c., and the order is not uncommonly reversed. This is evidently due to inconstant appreciation, although the task set was childishly easy. One of these patients asked if he might leave out the articles and conjunctions because he could then "do it better" if he wrote "cat, man" only.

Spontaneous writing, such as a letter to friends or relatives, is coherent and reasonable; but these patients complain, "I cannot pull it together very well yet," or, "If I'm writing I am apt to write on and then to wonder if I've put two l's or one; I'm not sure if I've written it wrongly or not." They have, however, a remarkable memory of the general contents; for instance, when No. 10 wrote a test letter to me nearly a year and a half after he had first come under my care, he began: "I have written you three letters, one about flouwers, one, a while later on, on same subject, and a third while I was at Northampton trying to tell you of my progress." This was entirely accurate; and yet he possessed no record by which he could have refreshed his memory.

These patients have no difficulty in reading aloud; when, however, they read silently, they are liable to miss the general sense of the passage, and to omit words or phrases essential to the argument. This tendency becomes particularly obvious if they are asked to write an account of what they have just read to themselves. For instance, I handed No. 5 his own story in print: "My name is Charles Hewitt and I live at Laurel Cottage, Pilley. Before I joined the Army I worked for Miss Drummond for nearly two years. I worked in the garden and looked after the pony. He was a forest pony, bay, with a dark mane and tail." This he read to himself and wrote, "I was worked for Miss Drummond and looked after a pony and Trap"; then he said, "That's all I remember." He was then asked to read it aloud, which he did perfectly and without hesitation; but all he could communicate in writing was as follows: "I worked for Miss Drummond and I looked after a pony and trap and helped in the garden." This loss of capacity was not based on any defect in the power of writing, for he copied his own story perfectly in cursive handwriting and was able to write a coherent letter to his relatives, mentioning the friends who had visited him by name. Here again symbols could be better utilized for spontaneous thought than for an intellectual effort made to command.

This is not due to want of education, for Lieutenant M. (No. 10) showed exactly the same disability. He read to himself the following passage from the newspaper: "News has been received from the Piraeus that the Greek Government proposes to proclaim martial law throughout Greece, and arrest M. Venizelos and his principal political friends." He at once wrote, "News received from Biraeus, that the Greek Government intend to proclaim martyal law throughon grace." He then read the paragraph aloud correctly, and asked to say what it contained replied, "News has been received from — — from — — that the Government of Greece intends to proclaim martial law throughout the country — — something wrong there — — because the political friends of Venizelos somewhere — —." His account died away in an unfinished sentence.

Drawing, even from a model, shows this want of consecutive memory and intention. These patients do not, as a rule, block out the drawing but tend to begin at some one point and follow round the outline of the object; this is also evident when they attempt to reproduce it from memory. Told to draw an elephant, No. 8 succeeded in producing a four-legged creature with no trunk or ears; but No. 10 and No. 5 could not form a coherent figure of any kind.

None of these patients could draw a plan of a room with which they were familiar. No. 8, who was an excellent draftsman before the injury, started well, but forgot the windows and the doors; moreover, he placed his seat alongside the fireplace, whereas it was in the middle of the room. He forgot the table in front of him but filled in several details, such as my weighing machine and typewriter, of little comparative importance.

The act of counting was perfect, but simple arithmetical operations were a trouble to all of them. They were subject to curious lapses, such as 6 + 8 = 10 in a simple addition sum, otherwise correctly carried out; sometimes an integer was carried over, sometimes it was forgotten. No. 10, who had been an accountant, could neither add nor subtract; he said, "I seem to get tangled up in the process." In every case coins were named correctly, but there was profound difficulty in stating the relation of any two of them to one another; for example, when a sixpence and a halfcrown were laid on the table, No. 10 said, "Let me see — — twenty-two — — I thought of twenty-four, but it's not"; and yet he gave many of the relative values correctly.

This tendency to confusion comes out perpetually in the operations of daily life. For instance, No. 8 complained, "When I'm going to shave I can't collect my things. I have to look hard at them all and then I am sure to miss some of them. In the same way I have to look at the things on the breakfast table. I see them all but I don't 'spot' them. When I want the salt or the pepper or a spoon I suddenly tumble to its presence. After my belt had been cleaned, the runners had gone back and I could not for the life of me think how to bring them into place." Patients belonging to the semantic group are much troubled by the movement of the streets, which puzzles and bothers them.

§ 5.—*Differences between these Various Forms of Disordered Speech contrasted.*

It has been universally recognized that the clinical manifestations differ greatly in individual cases of aphasia, and such differences have been attributed to a multitude of causes. I have attempted to show that they are produced by dissociation of a definite mental process, which I have called symbolic thinking and expression. They are not due to a loss of motor or sensory power, to destruction of images or to a diminution of general intellectual capacity, but are caused by the breaking up of one aspect of psychical activity analogous, on a higher level, to the sensory dissociations, which may follow a lesion of the post-central cortex. Certain physiological processes necessary for the normal exercise of the functions of language are disturbed by organic destruction of the brain. At first, in consequence of the widespread effects of this injury, the patient may be unable to speak, to read or to write. But as this state passes away all aspects of the disordered function may not be equally disturbed ; some actions are more easily performed and certain tests are carried out normally, whilst others are grossly affected. Such dissociation of symbolic thinking and expression is responsible for the clinical forms assumed by aphasia.

To each of these clinical forms I have given a name chosen to indicate its characteristic verbal defects. But it must be remembered that, although the power of using words shows the most extensive and gravest disturbance, other actions are affected which have nothing directly to do with such symbols. The name applied to each group of aphasic disorders is drawn from a grammatical source, because it is in the use of language that the changes are most evident and characteristic ; but the functions which suffer extend beyond the limits of verbalization.

Moreover it is well to remember that in each of these pathological groups both word formation and also the correct production of the phrase are affected. Thus in verbal aphasia words are evoked with difficulty and tend to be abnormal in structure. Nominal aphasia leads to defective use and understanding of words as names or indicators. Loss of syntactical power disturbs the internal balance of a word as an orderly rhythmic expression and so leads to jargon ; whilst semantic disorders interfere with capacity to comprehend and retain the general significance of a word as part of a complete act of language.

In the same way the orderly structure or intention of the phrase is affected in one way or another, whatever form the disturbance of symbolic thinking may assume. In verbal aphasia words cannot be evoked at will and retained so as to form parts of a perfectly constructed sentence. When the power of naming is affected, the nominal portions of the phrase are either absent or defective and may be replaced by gestures. But the structure of the phrase suffers most obviously with syntactical disorders. The normal rhythm of the sentence is destroyed, speech becomes syncopated, and those words which serve to bind together its constituent elements are absent or ineffective. Lastly, the patient who suffers from a semantic affection of speech pauses in his conversation, like a man who has lost the thread of what he wanted to say ; he falls back on expressions such as, "Well, you see it's like this," and sentences may die out unfinished.

All these disorders have both a formative and emissive aspect. In verbal aphasia the latter is the more prominent and obvious, but it must not be forgotten that there is also another side; these patients cannot retain with certainty a series of words or some unusual expression and this makes it difficult for them to read to themselves with ease and complete understanding. Moreover, the words uttered internally may be badly formed and so lead to confusion; No. 17 complained, "I can't read a book because I'm bothered, when I say the words to myself"; this comes out particularly clearly on attempting to write to dictation. With nominal aphasia the disturbance is evident both on attempting to name an object, and to carry out a command given orally or in print. Syntactical aphasia consists of a gross disorder of the emissive side of speech; but there is also difficulty in executing orders communicated by word of mouth. Spoken language is not perfectly understood, although comprehension of print may be undisturbed. In semantic affections the predominant feature consists in failure to receive a uniformly correct impression of the general significance of words or phrases; on the emissive side the defect appears as a loss of executive intention. This is particularly evident in drawing and in games such as billiards.

It must not be supposed that the various aspects of symbolic thinking and expression usually appear in these isolated forms. Most cases of aphasia, especially in the earlier stages, show evidence of widespread disorders of language. Many of these changes disappear with the passing away of neural shock; but the loss of function becomes confined to one of these groups in a few cases only. On the following tables I have put together those instances where the disturbance seemed to correspond most nearly to a dissociated affection of one aspect of symbolic thinking and expression, and I have attempted to show how the patients behaved to some of the tests. It is impossible on a general table to give an exact account of the nature of the responses, but the diagrams may help to make clearer the contrasting features of the different varieties of disordered speech. In all cases where the series of answers was in any way abnormal some indication is given in words of the form they assumed. Where the space is filled by a O the act was performed perfectly.

When aphasia is looked at from this point of view, it is obvious that no single test can be expected to reveal one form only. All the methods of examination I have described were designed to show how the patient behaves when a certain task is set before him in different ways. But, when the results obtained with such tests, in the various cases, are put together, certain conclusions emerge which can be summarized as follows:—

TABLE VII.—ARTICULATORY SPEECH.

	Word formation	Intonation and stress	Syntax	Repetition of words said by observer
Verbal.				
No. 4 ..	Great difficulty ..	Syncopated and broken	0	Good
No. 6 ..	Profound loss; at first dumb	Syncopated and broken	0	Good
No. 17 ..	Great difficulty ..	Syncopated and broken	0	Good
Nominal.				
No. 2 ..	Good except when doubtful of a name	0	0	Words perfect; content inaccurate
No. 7 ..	Extreme difficulty of expression	0	0	Slow; tends to change content
Syntactical.				
No. 14 ..	Jargon	Hurried and jerky	Grossly affected	Impossible
No. 15 ..	Jargon	Hurried and jerky	Grossly affected	Very bad; jargon
No. 13 ..	Jargon	Hurried and jerky	Grossly affected	Jargon
Semantic.				
No. 10 ..	0	0	0	0
No. 5 ..	0	0	0	0
No. 8 ..	0	0	0	0

TABLE VIII.—COMMON OBJECTS CHOSEN AND NAMED.

	Choosing object similar to one shown	Choosing object named aloud	Choosing object named in print	Naming object indicated	Writing name of object indicated	Duplicate placed in normal hand
Verbal.						
No. 4 ..	0	0	0	Slow; articulation defective	Spelling bad..	0
No. 6 ..	0	0	0	Slow	Slow	0
No. 17 ..	0	0	0	Slow; articulation defective	Writing and spelling bad	0
Nominal.						
No. 2 ..	0	Slow	Slow	Gross loss ..	Gross loss ..	0
No. 7 ..	0	Slow	Defective	Gross loss ..	Impossible ..	0
Syntactical.						
No. 14 ..	0	0	0	Incomprehensible jargon	Affected ..	0
No. 15 ..	0	0	0	Tendency to jargon; sense correct	—	0
No. 13 ..	0	0	0	0	0	0
Semantic.						
No. 10 ..	0	0	0	0	Grossly affected	0
No. 5 ..	0	0	0	0	Slow; badly spelt	0
No. 8 ..	0	0	0	0	0	0

TABLE IX.—CLOCK TESTS.

	Imitation	Oral command	Printed command	Telling time	Writing time
Verbal.					
No. 4 ..	O	O	O	Content correct; articulation bad	O
No. 6 ..	O	O	O	Same ..	O
No. 17 ..	O	O	O	Same ..	O
Nominal.					
No. 2 ..	O	Gross loss ..	Gross loss ..	Gross loss ..	Gross loss
No. 7 ..	O	Gross loss ..	Gross loss ..	Impossible ..	Gross loss
Syntactical.					
No. 14 ..	O	Definitely affected	O	Impossible ..	Correct in sense, extraordinary nomenclature
No. 15 ..	O	Slow; hesitating	O	Slow; hesitating	Same
No. 13 ..	O	O	Almost perfect	Slightly affected	O
Semantic.					
No. 10 ..	O	Gross confusion	Gross confusion	Some loss (later perfect)	Definite loss; confuses hands
No. 5 ..	O	Gross confusion	Confusion ..	O	Confused
No. 8 ..	Somewhat confused	Confused ..	Gross confusion	O	O

TABLE X.—HAND, EYE, AND EAR TESTS.

	Imitation, face to face	Imitation in mirror	Pictorial command	Pictorial command in mirror	Oral command	Printed command
Verbal.						
No. 4 ..	Affected ..	O	Affected ..	O	O	O
No. 6 ..	Affected ..	O	Affected ..	O	O	—
No. 17 ..	Slightly affected	O	Slightly affected	O	O	O
Nominal.						
No. 2 ..	Grossly affected	Not quite perfect	Grossly affected	Affected ..	Grossly affected	Grossly affected
No. 7 ..	Grossly affected	Very slightly affected	Grossly affected	Affected ..	Grossly affected	Grossly affected
Syntactical.						
No. 14 ..	Slow, but correct	O	Slow, but correct	O	Affected ..	O
No. 15 ..	Slow, but correct	O	Slow, defective	O	Affected ..	Affected
No. 13 ..	Affected ..	O	Affected ..	O	O	O
Semantic.						
No. 10 ..	Gross loss ..	Affected ..	Gross loss	Gross loss	Gross loss	Gross loss
No. 5 ..	Gross loss ..	Slightly affected	Gross loss	Affected ..	Affected ..	O
No. 8 ..	Affected ..	Slightly affected	Gross loss	Affected ..	O	O

(*a*) *Verbal aphasia*.—In severe forms of this disorder the patient's utterances may be reduced to "Yes" or "No," and even the words cannot always be evoked for voluntary use. As speech returns his vocabulary increases, his enunciation is slow and halting. Any word he is able to recall can, however, be used for naming an object; it may be so badly pronounced as to be scarcely recognizable, but it is applied correctly. When the patient attempts to repeat what has been said to him the articulatory sounds are imperfect, but he can usually utter more words than are possible spontaneously. It is characteristic of this form of aphasia that words are evoked with difficulty and tend to be abnormal in structure.

After the stage of neural shock has passed away, the power of choosing an object to oral or printed commands becomes perfect; for in this case the words he requires are presented to the patient by the observer. Even orders necessitating choice, such as the hand, eye and ear tests, can be carried out correctly if given in print or in words spoken aloud.

At first writing may be extremely difficult, or almost impossible. But as his spoken vocabulary increases the power of writing is regained, although to the last it tends to show the same errors as articulatory speech. These patients cannot spell, and find difficulty in remembering the order of the letters even in simple words. They write more easily to dictation, but are unable to carry in the memory a string of words or a long phrase. They can copy with ease printed matter in cursive handwriting.

The power of reading to themselves with enjoyment is spoilt by the difficulty in remembering a series of words accurately; they are frequently compelled to look back to the beginning of a long sentence in order to obtain its full meaning.

The verbal aspect of numerals is affected, but not their significance. Thus, when looking up the page of a book or scoring at cards the patient may utter the wrong number, but acts as if he had said the right one. Simple arithmetical operations can be carried out correctly, except in very severe cases; then it is not the process of addition or subtraction which is forgotten, but the act fails because of difficulty in remembering the requisite figures.

These patients can draw, play card games and enjoy jokes set out in print or in pictures. In fact, the disorder from which they suffer affects mainly verbal structure and words as integral parts of a phrase; their nominal value and significance are perfect.

(b) Nominal aphasia.—From the verbal point of view this is essentially a defective use of names. Not only does the patient fail in naming objects placed before him, but he has difficulty in employing expressions which give to words their value as a distinctive nominal indicator. For example, the only difference between "five minutes to eight" and "five minutes past eight" lies in the prepositions "to" and "past," and it is here that the patient is liable to fall into error rather than over the numbers five and eight.

When asked to point to an object named aloud or in print, the choice, if correct, is made slowly and with difficulty; for it is the nominal value of the word which is affected and not, as in verbal aphasia, its structural formation. This comes out strongly when the words are made to carry a command requiring choice, and the hand, eye and ear tests are badly executed to verbal orders given aloud or in print.

This inability to find correct names applies to the letters of the alphabet. The patient may be able to utter them in sequence to a varying extent, but he cannot be sure of naming isolated letters correctly. This greatly hampers his power of reading; for, when arrested by a word, he attempts to spell it letter by letter and is confused by the false names he is liable to give them.

Writing is gravely affected, and, although the patient can copy printed matter correctly in capitals, he may be unable to do so in cursive handwriting.

Repetition *viva voce* is perfect provided nothing further depends on the act; but writing to dictation, or any other action demanding choice, is performed with difficulty to spoken commands.

These patients can usually count, but suffer from defective appreciation of the value of single numbers. Thus No. 2 did not fully recognize the significance of such a number as 18 until he had counted up to it. This want of immediate comprehension of the nominal meaning of figures leads to want of ability to carry out simple arithmetical operations. This defect varies greatly in form and extent, but it was present to an obvious degree in my most highly educated patient (No. 2).

Games, such as cards, which demand rapid and correct recognition of names and the power to register a score, are impossible. No. 2 was, however, excellent at chess and could put together a puzzle without difficulty, whilst other patients of this group played draughts and dominoes with pleasure.

They could draw from a model or from memory after it was removed from sight; but, when asked to draw some such figure as that of an elephant, the result was extremely unsatisfactory. All the distinctive parts of the beast were usually omitted; and yet No. 2 made spontaneously excellent drawings of the animals he had shot before his injury.

These patients had no difficulty in appreciating the full significance of pictures and could understand jokes provided they were not conveyed in long and complicated phrases.

One of the most instructive forms assumed by this loss of function is the difficulty experienced by the patient in drawing a ground plan of some room with which he is familiar. However badly the verbal aphasic may draw, he succeeds in indicating the relative position of the windows, doors and principal pieces of furniture. But the patient with nominal aphasia fails to produce a correct plan, and usually slips away with an attempt to express the constituent parts in elevation; and yet if he is asked "Where is the table?" or "Where is the window?" he can usually point to their situation. A similar want of power to indicate relative positions appeared during the tests for naming common objects or colours. They were laid on the table in front of the patient and he soon became familiar with the position of each one of them. They were then hidden from his sight, and when a duplicate was shown to him he usually had no difficulty in pointing to the situation of the similar object, although he could no longer see it. But if the name only was given to him, either aloud or in print, he had difficulty in pointing to the relative position of the object on the table; for, given the word, he attempted to remember the names of the test objects in their order and became, in consequence, confused.

(c) *Syntactical aphasia.*—This is an easy form to distinguish because the patient talks jargon. The verbal aphasic speaks slowly and with difficulty; each word is produced with an effort. But with uncomplicated syntactical defects, speech is voluble and words are emitted with great rapidity. Sometimes each one is comprehensible, however difficult it may be to gather the full meaning of the phrase; but in other cases the words uttered are pure jargon. One of the most characteristic features of this form of speech is the want of grammatical coherence; the words tend to be ejected in a disconnected stream.

The power of naming objects placed before him may be retained by the patient in spite of the jargon by which he is hampered. Not infrequently, when he cannot utter a word or when the sound emitted is incomprehensible to his auditor, he can write the name correctly. In the lighter forms of this affection he may vary greatly in the expressions he uses for the same object, though each of them bears some distinct relation to its essential nature. Thus a pencil may be called at one time "black-lead" at another "blacking."

These patients can understand what they read to themselves provided they do not attempt to formulate it in words. Thus No. 14 read the daily papers and could demonstrate to me correctly the progress of the War by pointing to large scale military maps; but as soon as he tried to describe some event in words he became confused.

The difficulties experienced in writing show that internal speech is also disturbed by jargon. Single words, especially names in common use, can be written correctly, but any attempt to convey a formulated statement is liable to end in confusion. Patients suffering from the more severe degrees of this affection cannot write a letter, but in the lighter cases writing is easier than speech. They can all copy correctly and transcribe printed matter into cursive handwriting.

This disorder is essentially one of auditory balance and rhythm. Syntax, as the expression of this aspect of phrase formation, suffers greatly and, combined with the destruction of the internal balance of words, leads to jargon.

(*d*) *Semantic aphasia.*—So far the names I have applied to the various forms of disordered speech have borne some relation to the verbal defects. For this group, however, it is difficult to find a suitable designation that will express the essential nature of the disturbance, which extends beyond the limits of organized words. I have chosen the term "semantic" as a label for this form of aphasia because the affection comprises a want of recognition of the full significance or intention of words and phrases. But other functions suffer that have nothing to do directly with verbalization. The patient may be unable to appreciate the complete meaning of a picture although he recognizes all its details. He can carry out a manœuvre, where each action suggests the next, but is unable to do so if he is compelled to formulate it as a whole. Power to bear in mind the ultimate intention of the derived action is diminished and the patient has no firm recognition of the final goal of his efforts.

He has no difficulty in forming words and can repeat what is said to him. But if he is told some simple story and asked to reproduce it aloud or in writing, many of the essential elements are omitted; this also occurs to an even greater degree after he has read it to himself silently. He cannot retain that total conception of episodic sequence which is necessary for complete narration.

The clock-tests reveal the nature of this disorder in a striking manner. The patient confuses the two hands, does not know how to approach the task of setting them to verbal or printed command, and forgets the meaning of "past" and "to" the hour. Even the attempt to imitate directly on one clock the time set on another may lead to confusion and be carried out uncertainly. For whatever the test the patient is liable to misunderstand the intention of what he is asked to do. On the other hand, except in the gravest cases, he has little difficulty in telling the time provided he is allowed to keep the clock in front of him until he has given his answer.

Arithmetical operations become impossible or are carried out uncertainly and with difficulty. The patient may be unable to add or subtract because the mathematical process itself is incomprehensible.

He fails entirely to comprehend most jokes, especially if they demand the complete understanding of a picture and its printed legend. He cannot play cards games or put together puzzles, which confuse him greatly.

These semantic disorders interfere seriously with the actions of daily life and render the patient useless for any but the simplest employment; and yet his memory and intelligence may remain on a comparatively high general level. He does not forget people or places and can recall accurately events both recent and remote. Thus No. 10 was able spontaneously to remember the subject of the three separate letters he had written to me at various sittings during three years. But had I set him the task of repeating accurately a simple story, which he had been told or had read to himself, he would have become confused and many factors of importance would have been omitted.

CHAPTER V.—SYMBOLIC THINKING AND EXPRESSION.

Now that I have described the various dissociated forms assumed by disorders of speech in consequence of a unilateral lesion of the brain, it is possible to consider in detail the general nature of the functions which are affected. These I have grouped together under the title "Symbolic Thinking and Expression"; but I am anxious that this term should not be considered as defining the limits of the disturbance. I should have preferred to adopt some entirely neutral appellation, and to define its meaning by enumerating seriatim the various activities which are found to be affected.

To each of the partial forms of aphasia described in this paper a name has been given, indicating the characteristic disorder of speech, although the use of words is by no means the only function affected. These terms are not intended to define the limits of the disturbance, but serve simply as a memoria technica. In the same way I have combined the functions which are affected by a unilateral lesion of the brain under the general heading of "Symbolic Thinking and Expression," because, in the majority of instances, the gravest disturbance is shown in the use of such symbols as words and figures. Any mental activity is liable to suffer which demands the perfect reproduction of some symbol between its initiation and fulfilment. I do not, however, believe that it is possible to include within one categorical definition all the activities which experience shows to be affected; and yet from a physiological point of view they form a group as definite as those which underlie sensation.

Since it is impossible to define the exact limits of what I have called "symbolic thinking and expression," I shall enumerate the various actions comprised under this heading, which are affected by unilateral lesions of the brain. For the present I wish to exclude cases of bilateral affections, and all patients who showed evidence of definite destruction of sensory images. These must form the subject of an independent research; but they cannot be understood until the principles laid down in this paper have been clearly grasped.

It is not the "general intellectual capacity" which is disturbed by these lesions of the brain, but the mechanism by which certain aspects of mental activity are brought into play. Behaviour is affected in a specific manner; an action can be carried out in one way, but not in another. Thus, all these patients could choose from amongst a series of shapes or colours, the one that had been shown to them. They had no difficulty in picking out an object, which corresponded to a duplicate placed in the hand out of sight. All these are acts of direct matching; but the more definitely the task demands for its perfect execution symbolic formulation, the more certainly will it be badly executed.

The highest stage is reached in formulating a proposition, and, as Jackson pointed out, it is this form of mental expression which suffers most severely. Many patients who can choose an object correctly to oral or printed commands, can neither evoke the same spontaneously nor read it aloud; for both these actions require symbolic representation. If the word is given, it can be matched with some object on the table, but the adequate symbol cannot be called up at will.

When, however, the same patient is compelled not only to evoke a set of symbols, but to use them as a preliminary to action, as in the hand, eye and ear tests, he may be unable to execute oral or printed commands. For under such circumstances he must first formulate the proposition and then carry it into action correctly.

Any modification of the task, which lessens the necessity for symbolic representation, will render its performance easier. Thus a patient who finds extreme difficulty in imitating gestures, when sitting face to face, makes no mistakes when they are reflected in a mirror; for this is not a propositional act but one of direct imitation. If, however, he is asked to write down the movements visible in the mirror, he falls into the grossest errors because he is now obliged to formulate in verbal symbols what he sees.

The higher the propositional value of the mental act, the greater difficulty will it present. Thus a patient may execute a printed command to hold up his hand, although he is unable to carry out an order to touch with it his eye or his ear. The addition of the second factor has rendered the task too difficult; in the one case he appears to be able to read, whilst in the other he seems to suffer from "alexia." The larger the number of possible alternatives presented by the order, the more certainly will the desired action be defective. Thus it is possible by means of a series of printed cards to grade the severity of the patient's disability. He is first required to carry out a series of movements such as "lift your hand," "shut your eyes," &c., which name one part of the body only. Then the right or left hand is specified, and the task is gradually increased in severity until at last it is brought up to the multiple alternatives of the full hand, eye and ear tests. Somewhere on this ascending scale of difficulty the aphasic will break down, and this gives a rough indication of the extent of the loss of ability to carry out printed commands.

On this principle we can explain the behaviour of No. 2, who was unable to name colours when asked to do so, but never failed to describe them appropriately if he was allowed to use a simile or metaphorical phrase. For example, black was "what you do for the dead"; finally, during later observations at this stage, he was able to shorten this phrase to "dead." He could now name the colour by a single word "dead," because of its metaphorical significance, although he was unable to call up the more directly nominal expression "black."

The large majority of aphasic patients can copy printed matter into capital letters; this is purely imitative. But to copy it in cursive handwriting is an act of transliteration, demanding a certain degree of symbolic thinking. It is not surprising, therefore, to find that this form of activity is disturbed in some cases of aphasia.

It is comparatively easy to say the alphabet, the days of the week or the months of the year in sequence, provided it is possible to form the necessary words; long familiarity has made such tasks almost automatic. But to recognize immediately the significance of any single letter, day or month demands a higher order of symbolic thinking. Thus, a patient who can say such a series correctly may be unable to spell out the letters of a word or to comprehend the meaning of a definite date such as "Tuesday, the twentieth of March." He can set the clock at "4.45," but not when he is told to place the hands at "a quarter to 5." For in the first instance he places the short hand at 4, and then swings round the other consecutively up to forty-five minutes; in the latter he sets the hour hand at 5, and then stops, puzzled by doubt as to the nominal significance of the words "a quarter to."

The clock-tests also reveal the existence of symbols, which are neither expressed words nor numbers. The short and long hands have acquired a significance which converts each one of them into a direct symbol, and they are confused or used wrongly in many forms of aphasia (Table IX, p. 152). Moreover, we are in the habit of dividing up the space between any two numbers on the clock face into portions of an hour. If we are told to set half-past one, we not only bring the long hand opposite to the figure 6, but we bisect the space between 1 and 2, and place the hour hand in this position. The space between the two figures marking the hours has in itself a symbolic value. In many cases of aphasia this is affected, and the patient no longer sets the hour hand at a point proportionate to the position of the minute hand; he places the former opposite the figure 1, whilst the latter points to 6. Or, more confusing still, when told to set "a quarter to six," he may place the short hand at 6 and the long hand at 9, so that it is impossible to discover without questioning him whether he intended to set 6.45 or 5.45. Thus, not only the hands of the clock, but their relative position on the face have acquired a symbolic value, which is disturbed in many cases of aphasia.

The activity of the cerebral cortex is particularly associated with determination of the relations between external objects. Man developed the power of speech at a time when he had already acquired discriminative powers of a high order. The line along which the higher cerebral centres progressed was apparent in an increased capacity to distinguish variations of intensity, similarity, and difference, and spacial relationships. To this speech was added, which, apart from its emotional aspects, is concerned with expressing these relations. Definite symbols such as words and numbers were invented to register these attributes; but they are not the only relative factors, which may be disturbed in disorders of symbolic thinking and expression. So long as the patient has to choose an object from those in front of him, which corresponds to a duplicate shown to him or placed in his hand, he can remember the order in which they lie on the table even when they are hidden from his sight. But as soon as he is asked to formulate their order, that is, their relation to one another, he fails to do so. In the same way he may be unable to draw a plan of some room with which he is familiar, although he can recall and indicate correctly the situation of each single object.

In the same way No. 10 remembered that when buying tobacco he placed two shillings on the counter and received two ounces and threepence change; but he was unable to say how much it cost. He could register the facts correctly, but could not relate them to one another.

In the same way sense impressions and images can be appreciated and recalled, but the aphasic may have difficulty in expressing the relation between them. Thus, when tested with the compass-points, he may be entirely unable to answer correctly in speech or writing although he possesses the necessary words; and yet there is no reason to suppose that sensibility is affected. For if the figures 1 and 2 are written on a sheet of paper, he can indicate correctly whether he has been touched with one or two points. Under the ordinary conditions he is compelled to formulate his sensory impressions and to express their relation to some other condition. But when the test is carried out according to the second method, he has only to match his sensory image with one of two patterns in front of him.

Another form of activity, which is particularly disturbed in the semantic group of speech defects, is what may be called the ultimate intention of the symbol. These patients have no clear or certain conception of the goal of the action they are asked to perform. This is profoundly evident in all that concerns words and figures. But it is not the individual words in their primary sense which are gravely affected. No. 10 understood the words "summer" and "time," and also was well aware that "summer time" signified that the clocks were changed with the advent of summer. But he was entirely unable to say whether they were put forward or back, and tried in vain to work out the problem. In the same way these patients cannot add or subtract with certainty, because it is the processes of arithmetic which have been lost, and not the direct significance of figures.

This failure to formulate the intention or ultimate goal of a desired action leads to loss of capacity to perform tasks not directly associated with words or figures. Thus, when No. 10 was threading a quadrilateral frame for his bee-hives, he could carry out the operation if the action consisted in bringing the wire across from one side to another, and then back again through neighbouring holes; but as soon as he attempted to go from corner to corner he failed entirely. He could carry out a continuous action, but fell into difficulty when he was compelled by the discontinuity of the task to formulate his intention. In the same way a young officer, No. 8, was unable to put together his belt when the slides had been displaced.

In this chapter I have attempted shortly to bring together the various activities which are disturbed, when symbolic thinking and expression are affected. I believe that they form a group of mental processes, which can be defined at present by enumeration only. Some may object to the name I have selected; but this has been chosen solely because the disorder of these functions is most often and most profoundly manifested in the use of words and figures.

SUMMARY.

(1) The results set forth in this paper are based mainly on the investigation of young men suffering from gunshot wounds in various portions of the head (p. 90).

(2) Certain new serial methods of examination have been adopted; these make it possible to utilize the inconstant answers, characteristic of all cortical lesions, which are so confusing unless the examination is made in a systematic manner (pp. 89-107).

(3) These observations show that disorders in the use of language, due to an unilateral lesion of the brain, cannot be classed under the categories of isolated affections of speaking, reading or writing (pp. 108-111).

(4) They cannot be explained as due to destruction of images, "visual," "auditory" or "motor" (pp. 111-113).

On the other hand, "word-blindness," "mind-blindness," and what Jackson called "imperception," are all associated with more or less disturbance of the power to form images, complicated in some cases by those affections of language which form the subject of this paper.

(5) The "motor" aspect of these disorders of language is not solely an "anarthria" or high-grade articulatory defect. Careful examination shows that, in cases of so-called "motor" aphasia, not only external speech, but certain aspects of internal verbalization are affected. Thus the patient may find considerable difficulty in imitating movements made by the observer sitting face to face or in carrying out the same actions to pictorial command; and yet they may be perfectly executed, when reflected in a mirror. For in the first case some formulation in words is required, whilst in its second form the test is verbally an act of uncomplicated imitation. Moreover, writing tends to show faults in word formation of the same order as those of articulatory speech and spelling may be affected (pp. 113-115).

(6) I have grouped together the functions, which are affected in aphasia and kindred disorders of speech, under the heading of "symbolic thinking and expression." This name has been chosen because the gravest and most definite disturbance is to be found in the use of words, figures, and other symbols (pp. 115-119).

(7) But this term must not be supposed to define exactly the limits and extent of the actual loss of function, which can be discovered by examination. For not only may the power of using words and figures in speaking, reading and writing be affected, but there are other tasks which the patient cannot execute with certainty and correctness. He may be unable to formulate or draw a plan of the relative position of objects with which he is familiar, although he can indicate the site of each one of them individually. He mistakes the significance of the two hands of the clock and fails to recognize the proportionate value of the space between the figures of the hours. He can draw from a model, but may be unable to reproduce the form of an elephant to command. He fails to comprehend the full significance of a picture, although he recognizes the details of which it is composed. Any act is liable to suffer which requires for its perfect performance the antecedent formulation of the ultimate intention or goal towards which it is directed (pp. 157-162).

(8) The more nearly a symbolic action approximates to a proposition the greater difficulty will it present and the patient will probably fail to execute it correctly. The closer the task corresponds to matching two sensory patterns the less likely is it to be affected by these disorders of language. Highly complex symbolic acts suffer more gravely than those of a lower propositional value (p. 159).

(9) Under the influence of lesions situated in different parts of the brain the various functions comprised under "symbolic thinking and expression" may become dissociated. This is analogous to the effects produced on sensation by injuries to the cerebral cortex. Each of these dissociated forms represents a fraction of the complex and highly developed psychical process (pp. 148-157).

In most cases of aphasia two or more of these aspects of symbolic formulation are affected; but in order to comprehend the nature of the disordered functions it is necessary to select for examination patients in whom the disturbance is as nearly as possible confined to one of these forms of dissociation. Subsequently it is possible to understand the more complex aphasias and to enumerate in full the various actions comprised under symbolic thinking and expression.

(10) The various dissociated forms of symbolic thinking and expression may be comprised under the following headings:—

(*a*) Verbal Aphasia. This is essentially a defect of word formation. Words are evoked with difficulty and the vocabulary is greatly restricted. Enunciation is slow and halting. Writing tends to show the same sort of errors as articulatory speech, and spelling is defective. The patient has difficulty in reading to himself with pleasure, because he is unable to retain in his memory a long series of words. Numerals are affected to a slighter degree; their significance may be recognized and acted on correctly although they are wrongly enunciated. As speech returns, commands given in spoken or printed words can be executed; but orders which necessitate the evocation of some word or phrase may be carried out badly. These patients recognize, however, whether the task they are attempting has been performed correctly or not. They can draw, play card games, and comprehend jokes set out in print or in pictures (pp. 121-127).

(*b*) Nominal Aphasia. This is essentially a defective use of names and want of comprehension of the nominal value of words or other symbols. The patient reads with extreme difficulty, especially if he attempts to spell out the words. Writing is gravely affected and he may be unable to copy print into cursive handwriting. Writing to dictation and all actions demanding choice are performed with difficulty to spoken commands. Counting is possible to a varying extent, but the significance of numbers, the power to carry out simple arithmetical operations and appreciation of the relative value of money are usually more or less affected. The power to draw a strict ground plan of some familiar room is defective. These patients cannot play cards but chess and draughts may be possible (pp. 127-136).

(*c*) Syntactical Aphasia. This is an easy form to distinguish because the patient tends to talk jargon. Not only is articulation of the word ill-balanced, but the rhythm of the phrase is defective and there is want of grammatical coherence. These patients can read if they are not compelled to reproduce the meaning in words. Writing is usually less affected than external speech, although it tends to be disturbed by verbal jargon (pp. 136-142).

(*d*) Semantic Aphasia. This consists in a want of recognition of the full significance of words and phrases. The patient may understand each word or short phrase, exactly as he can comprehend the details of a picture; but the ultimate meaning escapes him. He fails to comprehend the final aim or goal of an action imposed on him from without. He cannot formulate symbolically a general conception, although he can enumerate the details of which it is composed. He can read and write, but the result tends to be inaccurate and confused Counting is possible and the value of numerals can be recognized, but appreciation of the nature of arithmetical process is defective. These patients cannot play games, and jokes set out in print or pictures are rarely apprehended in their full significance.

CONGENITAL FAMILIAL SPINAL MUSCULAR ATROPHIES AND THEIR RELATION TO AMYOTONIA CONGENITA.

BY KNUD H. KRABBE, M.D.

(From the Children's Department of the University Hospital (Rigs-hospitalet), and the Nerve-Polyclinic of the University Hospital, Copenhagen.)

SINCE Oppenheim, in 1901, described a disease which he called myatonia congenita (amyotonia congenita), accounts have been published of more than sixty cases of this disease, ten of these with autopsy. Some features of these cases are striking. One is that no case of amyotonia congenita in adults could be found in the literature. Possibly the persons who have had the disease are cured or dead. If the end result has been recovery it would conform well with the theory of Oppenheim and other authorities on the disease, that it is due to a retarded development of the muscles, and that the prognosis of the disease in all cases is good. This hypothesis conforms with Spiller's and Lereboullet-Badouin's findings at autopsies in some cases, where the spinal cord was found to be normal and only slight changes in the muscles were demonstrable. Spiller's case is, however, somewhat doubtful, as it was complicated by ocular phenomena and imbecility. Bing found normal muscle at a biopsy in a case of amyotonia congenita. However, this did not prove that all the muscles were normal. In eight other cases—Rothmann, Reyer-Helmholtz, Archangelsky-Abrikosoff, Marburg, Collier-Holmes, Griffith-Spiller, Laignel-Lavastine-Voisin, and Kaumheimer—considerable changes were found in the spinal cord and the muscles: atrophy of the anterior horn-cells, the anterior roots and the muscle fibres, in some cases also hypertrophic muscular fibres. These findings do not point to a retarded development, but rather to a much more profound change, and it might be supposed that these children, if they had lived, would have been affected by severe and irreparable atrophies and paralyses. Further, the histories of cases of amyotonia congenita show another problem; it is that of the heredo-familial nature of the disease. As is well known, in the overwhelming majority of cases no data pointing to heredity have been found, but in a few cases there has been a heredo-familial history. In the literature I have found four: the cases recorded by Beevor, Silvestri, Sorgente, and Skoog. This relation may only be explained in one of two ways.

One is, that amyotonia congenita is a heredo-familial disease conforming to Mendelian laws, but certain conditions must be present to determine its development. The other and more probable explanation is that the term amyotonia congenita really covers two different diseases, of which the one is heredo-familial, the other is not, a great difference existing between the heredo-familial and the non-familial diseases. The theory that the so-called amyotonia congenita represents two different diseases would also explain the other fact, that many patients are probably cured, while in other cases with an apparently less severe onset death has followed, and microscopy has shown extensive atrophy of muscle and disease of the anterior horn-cells.

If this last theory is correct, there exist two diseases: (1) Amyotonia congenita, which is characterized by a congenital hypotonia of the muscles, hyperflexibility of the joints, and slight pareses or pseudopareses, but no atrophies; this disease is not heredo-familial and the prognosis is comparatively good; only few cases (Spiller's and Lereboullet-Badouin's) have come to autopsy.[1] (2) A heredo-familial congenital muscle-disease, which bears some resemblance to amyotonia congenita, but shows, in addition to the hypotonia and hyperflexibility, marked atrophy of the muscles and is heredo-familial; in a certain number of these cases the heredo-familial factor cannot be directly shown as in the case of all the heredo-familial diseases; this last disease should show atrophy of the anterior horn-cells and of the muscle-fibres.

These theories are not alone supported by the above-mentioned considerations, but primarily by the following small series of cases.

The problem as to the relation between the myopathies and amyotonia congenita has been much discussed in the Neurological Section of the Royal Society of Medicine after lectures by Beevor, Batten, Collier and Homes, Fearnsides and Smith, Whait and Naish. Collier and Batten held antagonistic points of view. Collier did not see much clinical resemblance between amyotonia congenita and myopathy. He considered as the chief points of distinction the following: (1) The absence of any familial tendency. (2) The onset of the disease; it is either congenital or it appears rapidly after an acute illness. (3) The course of the disease is usually towards recovery. (4) The distribution of the muscular affection. (5) The invariable absence of neck. The patient did not try to lift her head, but turned it a little herself. The sterno-mastoid muscles seemed relatively powerful. The muscles of the back appeared to be somewhat atrophied. Respiration chiefly of the abdominal type, but also a little costal. Abdominal muscles seemed more feeble than normal. Abdominal reflexes could not be elicited. Reaction to pain on pricking. The chest somewhat narrow above, larger below. Sternum abnormally curved.

---

[1] As for the so-called acute forms of amyotonia congenita, I agree with Spiller and Batten, who consider them to be acute poliomyelitis, which undoubtedly may occur in the first months of life.

Lower extremities lay slightly flexed with normal abduction at the hip, slight flexion at the knee and very little supination at the ankle; position of the toes natural. Movements of the joints free, only abduction and flexion of the hips somewhat restricted. Tone diminished, no hyperflexibility (because of the atrophy?). Patellar, Achilles, and plantar reflexes could not be elicited. The condition of the skin and muscles resembled that of the arms. They gave the impression that bones with ill-developed muscles were loosely hung in a sack of skin and subcutaneous fat. The patient could move all her joints, but extension was slight and weak. No peroneal phenomenon.

The muscle reaction to the galvanic current was as follows: K.C.C.=5, A.C.C.=5·5, A.O.C.=10, K.O.C. > 10. The responses were small and slower than normal. Faradic examination could not be undertaken.

The patient was discharged after two days. Afterwards she was treated as an out-patient with massage and electricity. Some insignificant movements in the extremities resulted, but in February the child developed an acute febrile disease and died in a few days. The parents consented to an autopsy by me at the Anatomical-Pathological Institute of the University. (I record my indebtedness to Professor Dr. Johs. Fibiger for his permission.)

The brain showed nothing macroscopically abnormal, but the spinal cord was strikingly thin and firm throughout. Sections were taken from different parts of the central nervous system, the sciatic nerve, the femurs and muscles for microscopic examination. The muscles were thin, pale and covered with a thick layer of subcutaneous fat.

The sections were hardened in 4 per cent. formaldehyde; some of the hardened sections were later treated with Weigert's brown chromic acid stain; others with chromic acid-acetic acid mixture and imbedded in celloidin and the paraffinoid of Claudius. The following methods of staining were carried out: Nissl's toluidin-blue staining, Weigert-Kulschitzky-Wolter's method for medullary sheaths, Bielschowsky's staining, Herxheimer's staining of fat. The muscle-sections were stained with v. Gieson-Hansen's and Herxheimer's methods.

Microscopic examination.—In the sections from the cerebrum and cerebellum, the great ganglia and pons, nothing abnormal was found. The pia mater of the spinal cord was normal; no wasting of medulla was seen in the appropriately-stained pictures and no augmentation of the glia; only in the anterior root exit zones was there a diminution of the medullary fibres, and corresponding to it a slight augmentation of the drank well. Her crying suggested that she had a difficulty in "weeping through." No convulsions.

On November 27 she was admitted to the children's department of the Rigshospital (University Hospital) of Copenhagen.

She (fig. 2) was well nourished. The head dolichocephalic. Tension of the fontanelles normal. Nothing abnormal about the eyes or fauces. Stethoscopic examination normal. The Bordet-Wassermann test was negative. Weight 5,480 grm. Temperature normal. Urine no abnormal contents. Hæmoglobin (Sahli) about 80 per cent. The stools were normal. X-ray examination of the upper and the lower extremities showed no abnormality of the bones, but the shadows of the muscles were seen distinctly, and were indicative of great atrophy as compared with the muscles of a normal child.

The patient seemed to have normal intelligence: she did not cry or tremble.

Neurological examination.—The pupils reacted to light. Ophthalmoscopic examination negative. Eye movements free, no ptosis, nystagmus nor strabismus. Right palpebral fissure somewhat more open than the left, especially on crying. Slight facial paresis on the right side. Chvostek's sign absent. The expression of her face, the smiling, crying and mimicry movements were natural. Facial and tongue muscles did not seem atrophic, but the temples were markedly sunken on both sides (atrophy of the temporal muscles?). The tongue was protruded incessantly and exhibited marked fibrillary movements.

Upper extremities.—Shoulder-joint abduction of 90 per cent.; outward rotation of humerus, flexion of the elbow-joint; marked pronation of the forearms; wrists slightly flexed; the four ulnar fingers slightly flexed; thumb hyperextended and very abducted. Movements of all the joints free, only supination and extension of the forearms somewhat limited. Hyperflexibility of the finger-joints, not of the other joints. Palpation of the arms gave the impression that the skin and subcutaneous tissues formed a very thick and easily displaced layer over atrophic muscles. The skin of the upper arm showed a very deep fold on the flexor aspect.

The patient could move her arms, apparently using all the muscles except the supinators of the forearms; all the movements were rather slow with little excursion; her hands seemed to be most active, but she was not inclined to grasp anything put in her hand. No tremor, no choreiform or athetotic movements. Pricks everywhere on the arms made her cry a little, but did not evoke defensive movements. Active vaso-motor reaction round the pricks. Tendon reflexes could not be obtained, nor the reflexes of Léri, although the patient showed reaction to pain. Tone much decreased. No cyanosis, œdema or other vaso-motor disturbances. No Trousseau tetany-phenomena.

The trunk.—When the child was placed erect, there was marked lordosis of the cervical spine and arcuate kyphosis of the back. No stiffness of the before death. Immediately after birth, the mother noticed that she did not move her legs at all and her arms only a little. She could only draw the back of her hand up to her mouth, but apart from this movement her hand

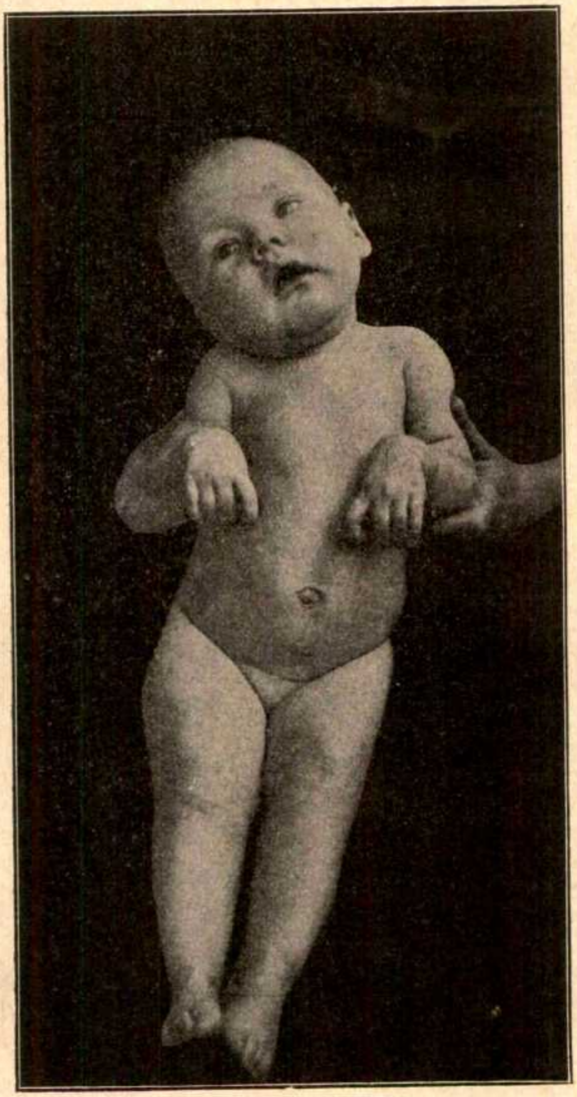


FIG. 2.--Case 2, C.

hung flaccid. Later, the mother noticed that the child could move her fingers and toes a little, and draw her legs up somewhat when she was raised. She could turn her head, but it dangled when she was made to sit up. She seemed to know her mother; when she got a little older, she laughed, babbled and paralysis. The arms lay naturally, were moved freely but feebly. The patient could grasp and hold objects put into his hands, but when upright he could not lift his arms above the level of his shoulders. Considerable loss of tone, hyperflexibility about the shoulders, but not about elbows or, scarcely, about the wrists. Tendon reflexes were not obtainable. The patient reacted to pin pricks. The muscles of the arms were atrophic, but covered by a thick layer of subcutaneous fat.

The body.—Chest normal. Abdominal reflexes present on both sides. The spine straight, not rigid. When the patient sat straight up and somewhat bent forward, he was able to hold his head up, but as soon as he was pushed somewhat back, his head fell backwards, and he could not lift it himself (fig. 1). The patient reacted to pin pricks everywhere.

Lower extremities.—The muscles of thighs and legs seemed atrophic but covered with fat. Loss of tone, but no hyperflexibility. The patient could not stand on his feet, he fell on his kness and hips when he was propped up. The limbs were moved feebly but without striking slowness.

Knee-jerk and ankle-jerk could not be obtained. Plantar reflex normal. The patient reacted to pin-pricks on the legs. He moved his limbs without ataxia or tremor.

The examination did not otherwise show anything abnormal; no sign of rickets. The testicles had descended, there was no enlargement of the abdominal organs, and the stethoscopic examination was negative. Skiagrams of the extremities showed the muscles to be feebly developed, and the spaces between them seemed more pronounced than normal (intermuscular fat ?).

No doubt this was a case of congenital familial progressive muscular dystrophy, and it is striking that the histories of the two brothers were similar. These cases closely resembled those described as amyotonia congenita. They were distinguished by (1) familial occurrence and (2) the fact that the disease does not improve, but that there is (in spite of the growth and development of the child) stagnation, relative retrogression.

While this case, like Wimmer's, was diagnosed as congenital familial muscular dystrophy, I propose mentioning some cases, first diagnosed as amyotonia congenita, later, in the light of additional facts, as heredo-familial disease, i.e., not amyotonia congenita.

Case 2.—A girl, born in August, 1914, died half a year old. The parents and three older sisters and brothers were well. The mother felt fœtal movements at the usual time and during the rest of the pregnancy. The child was born at term, labour was normal, and she was not asphyxiated. She was suckled by her mother for the whole of her life, and, apart from the disease of the nervous and muscular systems, she showed no symptoms till a few days

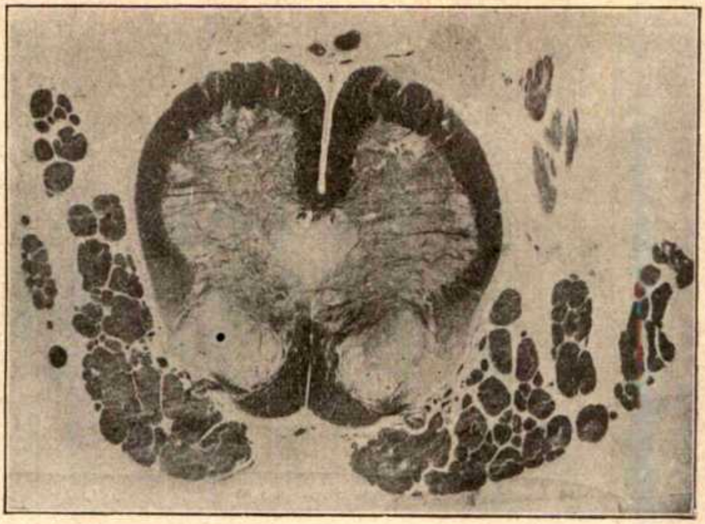


FIG. 3.—Spinal cord, stained with Weigert-Kulschitzky-Wolters method. Shows the degeneration of the anterior roots.

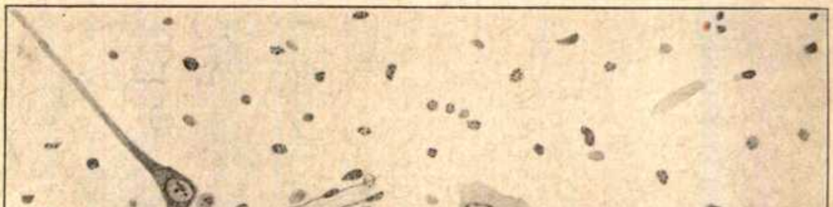


FIG. 4.—Anterior horn from the spinal cord of Case 2. Nissl staining.

glia. The anterior roots were very markedly degenerated; on the medullary-stained pictures they were white, while the posterior roots showed normal dark colour (fig. 3). The *nerve-cells*: In the anterior horns only a few of the great motor-cells showed normal size and form (fig. 4). Most of them were very shrunken, the protoplasm was less than normal, more angular and with a scanty number of prolongations. The protoplasm of these cells was ordinarily pale and homogeneous, without Nissl's bodies; in a few cases it was intensively stained, the cells showing a characteristic pyknoid condition. The protoplasm presented only a few vacuoles and there were no fat or pigment granules; the sections stained with acid fuchsin-vert-lumière showed a number of cells filled with acid fuchsin-stained granules. This observation must be judged with reserve; possibly these granules might be artificial. The nuclei of the cells were usually diminished, but less so than the cells as a whole; the nuclei were often eccentric. The chromatin of the nuclei was somewhat abnormal; it was distributed in comparatively large and intensely stained granules. Besides the nucleoli there were some small granules, stained in the same manner, perhaps expelled from the nucleoli. In some of the cells the nucleus appeared to be destroyed—a result, perhaps, of the preparation of the section. In a single nerve-cell there were two nuclei. In the most normal nerve-cells the Nissl bodies were preserved round the nucleus and destroyed in the peripheral part of the cell. The axis cylinders were relatively intensely stained and could be followed a long distance from the nerve-cells.

In each of the 10 μ sections there could be found only 1-2 relatively normal anterior horn cells; but in Clarke's columns the cells were normal, only with slight change of Nissl's bodies in the periphery; there were no shrunken forms.

The neuro-fibril sections showed well preserved neuro-fibrils in the best preserved nerve cells; in all the small and shrunken cells the neuro-fibrils were destroyed and appeared only as small granules.

While the amount of the glia in the white matter (seen apart from the anterior root zone) was absolutely normal, there was a considerable increase of glia in the grey matter. There was a greater number of glia-cells than normal in both the anterior horns, and there was a great amount of glia-fibres passing through the anterior horns in all directions, most dense in the anterior part, but otherwise evenly distributed in the anterior horns. There was nowhere any dense accumulation of glia. The glia-cells were nearly all of the fibrillary type; no ameboid, vacuolized or giant forms were seen. There was no increase of the satellite cells round the nerve-cells; medullary fibres and glia were evenly distributed throughout the spinal cord.

In the fat-stained sections no fat or lipoid granules were seen in the glia-cells or nerve-cells.

The vessels were everywhere normal; there was nowhere any perivascular infiltration with lymphocytes, leucocytes or plasma-cells. There seemed, however, in the anterior horns, to be an increase in the number of small vessels.

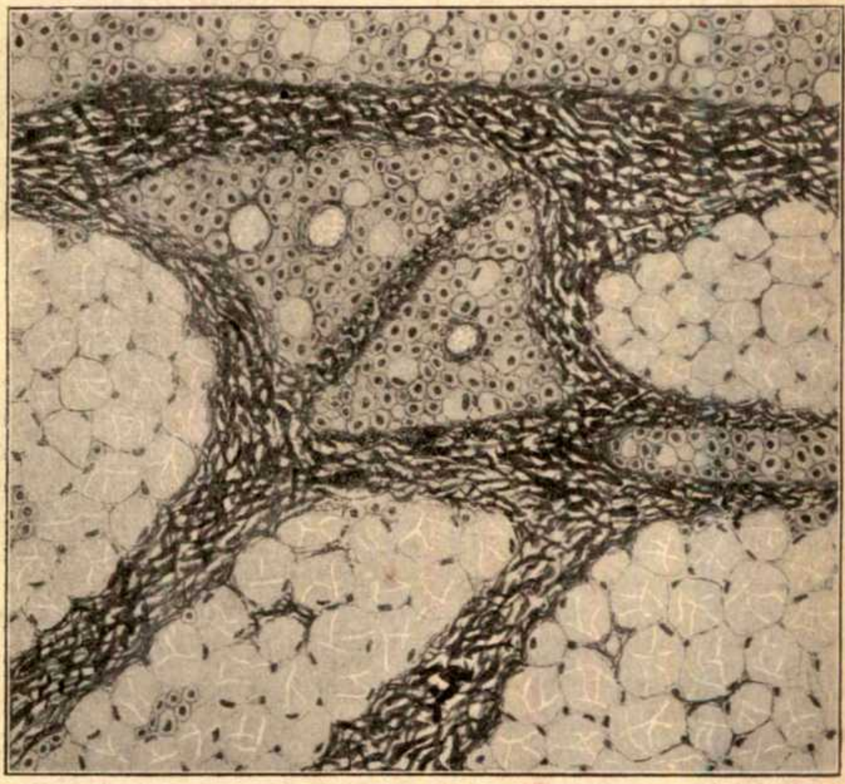


FIG. 5.—Muscle from Case 2, stained with v. Gieson-Hansen's method, showing normal and atrophic muscle fibres and connective tissues.

The above-mentioned section of the sciatic nerve showed a moderate diffuse increase of the connective tissue but no sign of inflammation.

The appearance of the muscle examined was characteristic (fig. 5): Some of its fibres were perfectly normal; but a considerable number of muscle fibres were very thin, without obvious striation and with a single central nucleus in every fibre. There were no hypertrophic fibres, but here and there transitory forms between atrophic and normal. In these transitory forms the nuclei were somewhat eccentric. The protoplasm in the atrophic fibres usually stained more darkly than that of the normal ones, and no transverse or longitudinal markings were seen.

The normal and atrophic fibres were separately distributed ; here and there, however, small groups of atrophic cells were seen in the normal bundles and normal cells in the atrophic bundles. Between the bundles there was much connective tissue. But there was no increase of the perimysium between the single fibres, no increase of the vessels, no sign of inflammation, no leucocytes. The atrophic fibre-bundles contained many more nuclei than the normal bundles. This increase of nuclei was, however, only relative, a consequence of the atrophy of the protoplasm. The transverse sections of the muscles showed the atrophic muscle-fibres to be far more numerous than the normal.

The clinical and post-mortem findings established the diagnosis: amyotonia congenita. It was a case of congenital muscular weakness with hypotonia of the muscles and loss of the tendon reflexes. The fact that there was atrophy did not tell against the diagnosis, for in many of the recorded cases of amyotonia congenita there had been atrophies. The post-mortem findings absolutely coincided with the cases of Rothmann, Archangelsky-Abrikosoff and Marburg, only the absence of hypertrophic fibres distinguished it from the cases of Badouin, Reyer-Helmholtz, Collier-Holmes, Griffith-Spiller, Laignel-Lavastine-Voisin and Kaumheimer. There was no history of heredity ; the three elder sisters and brothers of the patient were well.

Now, however, five years later, it appears that the disease was nevertheless familial ; in August, 1919, the mother applied to the polyclinic for children at the Rigshospital concerning a younger brother, whose history is as follows :—

Case 3.—M. J. C., born March 25, 1919, son of a coffee merchant. The three elder sisters and brothers were well. Apart from the dead sister there was no history of nervous disease in the family.

The mother had suffered from slight influenza during the first month of pregnancy, which, however, was normal. She felt fœtal movements at the normal time and during the rest of her pregnancy.

The patient was born normally at term ; his weight at birth was 3,500 grm. During the first five months he was suckled, but after that time he was given milk, milk-soup and gruel. His growth was normal.

The mother noticed soon after his birth that the child moved his legs very little and was absolutely flaccid. This flaccidity did not disappear: when he was about half a year old he could not lift his head from the pillow, but only turn it a little. He moved his arms freely, but not as actively as a normal child; he could grasp different things, but hold them with difficulty. He could lift his legs under himself and move his head from his pillow. There was no history of convulsions or fever. His mental development corresponded with his age, he was interested in his surroundings, smiled and was quiet and amiable.

At the hospital the patient was seen to be well developed and nourished, not anæmic. No glandular swelling of the neck, no swelling of the thyroid gland. Weight 6,750 grm.; length 64 cm. The fontanelles 2 by 2 cm.; teeth 0.0. Urine normal. V. Pirquet and Bordet-Wassermann reactions negative. Genital organs normal. The child had blue eyes, light hair, and was dolichocephalic.

Ophthalmoscopic picture normal. Pupils reacted well to light. Eye movements free. No strabismus, ptosis or nystagmus. The child followed moving objects with his eyes and turned his head in response to noise. No facial paralysis. No Chvostek's sign. He smiled sometimes; his expression was natural. Considerable fibrillary movements of his tongue. No paresis of the soft palate.

He could move his head from side to side but never tried to lift it from the pillow. When he sat semi-erect, there was lordosis of the cervical spine, so that his head sank between his shoulders. Though it tended to fall forwards or backwards, the child tried to keep it straight. The dorsal and lumbar spine showed a slight arcuate kyphosis. Respiration was mainly abdominal, only a little thoracic. The abdominal muscles were weak, and the child was unable to contract them. Abdominal and cremasteric reflexes could not be elicited. Only a little pain-reaction on pricking the trunk.

Upper extremities.—Position normal. No atrophy was seen, but on palpation through the thick layer of fat the muscles seemed atrophic. The child moved his limbs freely at all the joints, but the movements were weak. Tone seemed diminished, but there was no hyperflexibility at the joints. Tendon reflexes could not be elicited. He reacted to pricks with strikingly small movements and a little grunt, but no crying.

Lower extremities lay rotated outwards, a little flexed at the knees. There was *pes planus* on both sides. The foot formed a rectangle at the ankle with the heels prominent. The child was able feebly to flex and extend his lower limbs at the ankles and toe-joints. Very feeble flexion and extension at the knee, slight movement at the hip. When the leg was lifted, it fell immediately flaccid. When the child was propped upright, his knees gave way and he made no attempt to straighten them. Tone of the leg muscles was scarcely diminished, but there was hypotonia of the hip- and thigh-muscles. No hyperflexibility, but it was rather difficult to move the extremities outwards. Patellar and Achilles reflexes could not be elicited. Plantar reflexes of normal type but feeble. The child reacted by crying when the lower extremities were pricked.

Electrical reactions: K.O.C. = 8; A.O.C. = 5; A.C.C. = 2·5; K.C.C. = 4.

No doubt this boy suffered from the same disease as his dead sister. The pseudo-heredity of congenital syphilis could certainly be excluded. We must, therefore, diagnose these two as cases of familial congenital muscular dystrophy, which in no way differ from Wimmer's and my two cases of congenital muscular dystrophy. Only the progress of the palsies and the atrophies was more marked in Wimmer's case than in the two cases described here.

I have examined some more cases, diagnosed as amyotonia congenita, in the children's department of the Rigshospital. Further examinations of the family histories showed that the disease was familial.

Case 4.—N. H. B., son of a farmer, born October 7, 1915, treated in the children's department of the Rigshospital, January 19 to February 6, 1916.

No case of muscular or nervous disease in the family (cf. *infra*). The parents were well, an elder brother was sane (for description of the family, see figs. 6-7). The father's brother had disseminated sclerosis (the diagnosis certified at the Rigshospital's clinic for nervous diseases).

The child was born naturally at term. The mother had felt fœtal movements during the last half of pregnancy. The child was suckled by its mother during the first three months, afterwards he was given boiled milk.

The mother had noticed that the child since birth always held its hands in a certain position, flexed at the wrists, extended at the metacarpo-phalangeal joints and flexed at the interphalangeal joints. He could move his arms well and lift them, and he could move his fingers, but not straighten them out. There was also a somewhat abnormal position of the left foot. Further, during respiration, there was always marked retraction of the thorax round the ensiform process. The mental condition was normal.

On admission to the Rigshospital the child was small and flaccid, somewhat apathetic during the examination, and only on inspection of his throat did he cry a little. He was fairly well nourished. Weight 4,750 grm. The fontanelles 3 by 3 cm., somewhat sunken. No teeth. Stethoscopic examination normal. v. Pirquet's reaction negative. The pupils reacted to light. No atrophy of the facial muscles. When crying there was some tendency to draw the mouth to the left; there was, however, no marked facial paralysis. No Chvostek's sign. Fauces normal.

He could not lift his head from the pillow nor move it from side to side. When he was placed erect his head fell flaccidly backwards. The neck muscles seemed atrophic. Respiration chiefly abdominal; during respiration the lower part of the sternum was drawn deeply in and from it a deep sulcus was continued to both sides of the costal arch. Abdomen somewhat prominent, especially the epigastrium.

Upper extremities.—Right upper arm was abducted (about 90°), the forearm flexed to 90° and permanently pronated. Hyperextension at the metacarpo-phalangeal joints. The fingers were flexed. Left upper arm also somewhat

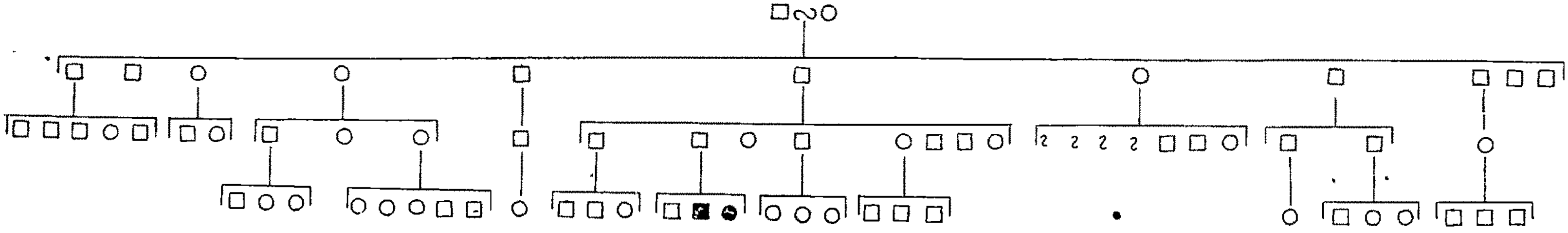


Fig. 6.—Case 4, father's family.

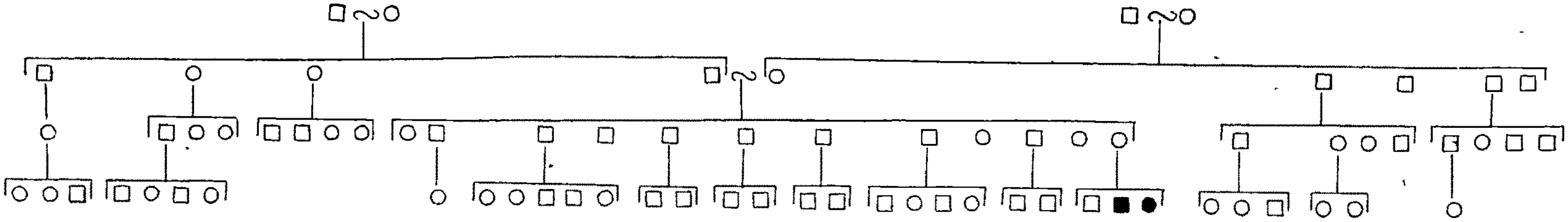


Fig. 7.—Case 4, mother's family.

abducted, but less than the right; the forearm flexed and pronated. Hand and fingers as on the right side. Passive movements could be executed normally. The patient held the left elbow a trifle flexed, rotating the right forearm and moving the right shoulder only a little. The muscles did not seem to be strikingly atrophic, but it was difficult to judge because of the abundant subcutaneous fat. No radial, nor Trousseau's phenomenon.

Lower extremities lay a little abducted and rotated outwards. Left foot lay in a slight valgus position; right foot had an arched instep and was rather in an equinus position. After trying to rotate the thigh inwards, the limb fell back to its original position. Passive extension at the hip was normal, there was no hyperflexibility. The knee could only be moved about an angle of 90°; the ankle could be moved freely. The patient could not himself lift his limbs from the bed or bend his knees, and he could only flex his ankles a little. The trophic condition of the muscles was difficult to judge because of the abundant fat. Sense of pain scarcely diminished. Patellar and Achilles reflexes could not be elicited. Plantar reflexes very feeble, of normal type.

Electrical examination of the left median nerve showed: K.C.C. = 6·5; A.C.C. = 4·5; A.O.C. = 10; K.O.C. = 10.

The contractions were very slow and feeble. The left peroneal nerve: K.C.C. = 6; A.C.C. = ? (10): A.O.C. = 13; K.O.C. = 13.

X-ray examination in the upper and lower extremities showed nothing abnormal in the bones. The muscles, especially the crural muscles, seemed somewhat feebly developed: the atrophy was, however, not very marked.

After the patient had been 14 days in hospital signs of pneumonia on the left side suddenly appeared, and death occurred after two days. An autopsy was not permitted.

The diagnosis was: Amyotonia congenita. As I had ascertained that Case 2, which was also diagnosed as amyotonia congenita, was really one of hereditary disease, I searched for further data about the family of this case from an uncle who was a doctor. He gave me the surprising information that the mother of the child two years after the death of the patient had a daughter, who a short time after birth had showed a paralysis just like that in the patient here described; the child died half-a-year old of pneumonia. After that there is no doubt that the disease, tentatively diagnosed as amyotonia congenita, was really the same congenital familial muscular dystrophy as in Cases 1, 2 and 3.

The following case showed the same clinical and pathological picture as that already described. The familial occurrence is here somewhat more doubtful.

Case 5.—G. S., daughter of a farmer, born April 6, 1918, admitted to the children's department of the Rigshospital, July 19, 1918; died there August 6,

1918. A sister of the father's father had from birth been paralysed in arms and legs, was wheeled about in a Bath chair, died about 8 years old. Otherwise no cases of paralysis in the family. The patient had no sisters or brothers.

The child was born in breech-presentation by extraction after version. She was given both mother's milk and tapioca-soup and milk. Even from birth the muscles of the neck and the extremities were flaccid, she could move her arms and legs very little and not extend them at all. She moved her fingers and toes quite well. The left arm was moved somewhat better than the right. The head could be moved only very slightly from side to side. The movements of the lower extremities improved, and by and by the patient could draw them somewhat up and extend them again. Never cramps or convulsions. The child was always active and happy and drank well.

The examination on admission showed the child to be mentally well developed. Expression of the face normal Cried vigorously. She was well covered with fat. Stethoscopic examination negative. The fontanelles 4 by 3 cm , tension normal. The cranium firm.

The temporal muscles seemed slightly atrophic. Fibrillary contraction of the tongue. Cervical muscles atrophic. When she lay down she could move her head only a little from side to side. When she sat, her head fell forwards or backwards. Thorax paralytic, the respiration only of abdominal type. Abdomen soft.

Upper extremities.—Shoulder and pectoral muscles atrophic, the other muscles seemed also atrophic, but less so; there was some difficulty in palpating through the subcutaneous fat. The muscles were flaccid. Considerable hyperflexibility at the shoulder-joints, not at the other joints. Forearms pronated. The patient moved the left arm better than the right. This could only be moved about ten degrees at the elbow and hardly at all at the shoulder. Left arm could be lifted a little from the bed. The power of the hands was somewhat better, the patient could hold a finger feebly. Reflexes could not be elicited.

Lower extremities.—The muscles were flaccid and seemed atrophic, but their condition was somewhat difficult to judge because of the abundant fat. The child moved her legs a little better than her arms, best at the ankles and toes, but very feebly. Reflexes could not be elicited. The sense of pain was everywhere normal.

X-ray examination of the extremities showed the muscles of all the extremities, chiefly those of the arms, to be somewhat atrophic. Bordet-Wassermann reaction negative. v. Pirquet reaction very slightly positive. The urine contained no sugar.

In hospital the child drank well at first, cried somewhat, but was otherwise rather quiet. After ten days pneumonia developed on the right side. Her respiration became short and shallow, and she coughed without expectorating. In spite of treatment with steam, camphor, and digalen injections, the patient collapsed and died August 6, 1918. At the autopsy broncho-pneumonia and purulent bronchitis of both lungs were found.

I examined the spinal cord microscopically, but not the muscles. The microscopic examination showed changes corresponding closely with those of my former case. In the different segments of the spinal cord a few of the anterior horn cells were normal, with a beautiful tigroid staining. But most of the anterior horn cells were atrophic, angular, and showed rubbed-out tigroid staining, the protoplasm being homogeneous or very finely granulated. Nuclei and nucleoli were, however, normal. The cells of Clarke's column were relatively more normal, as they were not diminished in size; they showed only in the periphery normal Nissl-granules; all the part round the nucleus was homogeneous. In the medullary-stained preparations the anterior roots were conspicuously degenerated, but the posterior roots retained their normal colour. In other respects they showed no degeneration of the white substance.

Pathologically this case resembles the first described, clinically also the resemblance is great. The familial history is somewhat more dubious, in that the paternal aunt mentioned might have been paralysed from a poliomyelitis or other disease.

In the following case the familial condition was not found in a brother or a sister, but in other, more distant relatives. The diagnosis, therefore, was dubious: either it was amyotonia congenita or congenital progressive muscular atrophy. Clinically the case was like the others.

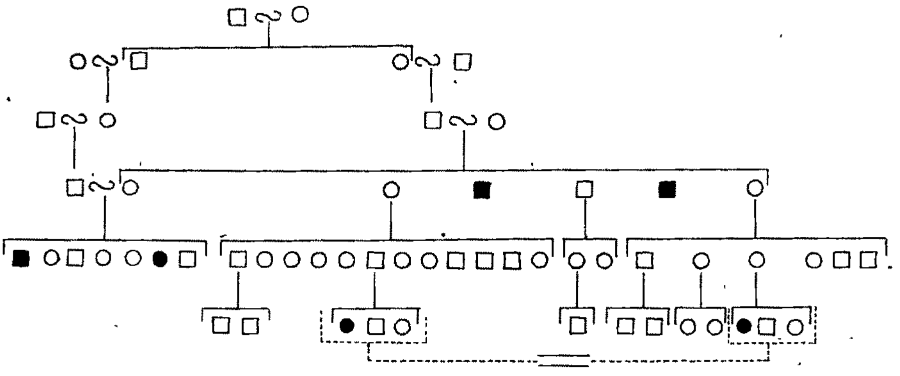


FIG. 8.—Case 6, father's and mother's family.

Case 6.—E. B. C., daughter of a lawyer, born April 12, 1917, treated in the children's department of the Rigshospital, July 10 to July 21, 1917.

The mother's mother gave me the following information: The parents were cousins. The two brothers of the mother's mother (and the father's mother) died about 8 months old; they were said to have been paralysed from birth, one of them in the upper, the other in the lower limbs. Further information could not be obtained, as they died fifteen years before the birth of the mother's mother. The father's mother's and the mother's mother's elder sister was related to her husband (fig. 8). She had had a son and a daughter, both of whom had been paralysed shortly after birth and had died suddenly about 8 months old in connection with catarrhal infections.

The mother was well during pregnancy, and had certainly not shown any sign of poliomyelitis. She felt fœtal life during the last part of pregnancy. Labour was normal, lasting only three hours. The weight of the child at birth was 3,500 grammes.

When the child was one month and a half old, the grandmother remarked that her legs were very flaccid. About fourteen days later the grandmother remarked that her arms were singularly abducted and flexed at the elbows, and that the flaccidness of the legs was not marked. The condition seemed, however, again to improve a little. She was suckled by her mother during the first month and a half, after that milk and barley water were given.

On admission to hospital the child was seen to be small and very flaccid, but well nourished. Her head was dolichocephalic, her fontanelles normal. Her intelligence seemed normal.

Pupils reacted well to light. Ophthalmoscopic examination negative. Eye movements free, now and then a little strabismus, no ptosis or nystagmus. Temporal muscles somewhat flattened. No facial paralysis. No Chvostek's sign. Facial expression smiling, and mimicry natural. The tongue did not deviate on protrusion, but it showed some fibrillary movements. Sucking energetic.

Upper extremities usually abducted to 90°, flexed at the elbows and rotated outwards. Wrists were slightly flexed. Free passive movements at all the joints, hyperflexibility in them all, especially the shoulders. The muscles, on palpation through the abundant fat, seemed very atrophic. The patient moved the elbows, both spontaneously and when pricked. The range of the feeble movements at the shoulders was limited. No tendency to grasp things. No tremor, athetosis or choreiform movements. Sensibility seemed everywhere diminished, the child did not cry even when pricked deeply, but did so when the wrist was flexed roughly. No vaso-motor reaction about the pin pricks. Tendon reflexes could not be elicited.

Trunk.—When the patient lay down, she only moved her head from side to side, but did not try to lift it. When she sat up somewhat, she could not hold her head up, it fell forwards or backwards. In spite of this she tried to lift it up. Muscles of the back were very flaccid; when she was set up there was a somewhat arcuate kyphosis of the dorsal and lumbar spine and lordosis of the cervical spine, so that her head sank down between her shoulders. Her respiration was chiefly abdominal. Abdominal reflexes could not be elicited.

No pain reaction to pricks on the body. No vaso-motor reaction. There was incontinence of urine.

Lower extremities.—Position practically normal. Tone everywhere diminished, and there seemed to be some hyperflexibility. Tendon reflexes absent. By pricking the sole of the foot the patient reacted both by contraction of the quadriceps and by defensive movements of the foot. She could actively but feebly move most of her joints.

Lumbar puncture yielded blood-stained fluid. Both this and the blood showed a negative Bordet-Wassermann reaction.

Electrical reactions: K.C.C. = 4, A.C.C. = 5·5, A.O.C > 10, K.O.C. > 10.

In hospital the child appeared mentally normal, smiled and babbled.

After ten days she was sent home, where she died after a short time. No autopsy.

A résumé of the cases and of Wimmer's case shows the following points:—

Familial incidence occurred in six cases undoubtedly, and possibly in one case. In two cases, Wimmer's patient and his brother, the mother had not felt fœtal life or only very slightly; in three other cases the mother felt life in a normal manner in due time. In one case the child was born in breech presentation by extraction after version; in all other cases birth was normal. In five cases the disease was noticed immediately after birth, in the sixth it was noticed about fourteen days later, and in the seventh one month and a half after birth. It is, however, very probable that the disease in these two cases also was congenital, but was not detected at once.

The pareses were extensive in all the patients. In one of the patients there was a trace of facial paresis; the cranial muscles, especially the eye muscles, seemed otherwise to have been spared. As a rule, the upper and lower extremities were affected in similar degree and more centrally than peripherally. The trunk muscles were also usually affected, all the children collapsed when propped up, the head fell forwards or backwards, and none of the children could lift the head from the pillow. The respiration was mainly abdominal, in several of the children the thorax seems to have been completely paralysed.

Tone was diminished as in amyotonia congenita, but the hyperflexibility of the joints, which is common in amyotonia, was observed only in the fingers and wrists and, in two cases, in the shoulder joint. The trophic condition of the muscles was often difficult to judge because of the abundant fat. Both palpation and X-ray examination showed great and extensive muscular atrophies. Fibrillary contractions of the tongue were observed in three patients only. Tendon reflexes were absent in all. The sensory phenomena were variable, as a rule the sense of pain was intact; in two of the patients, however, it showed a diffuse limitation. None of the patients showed any sign of syphilis.

The pathological findings in two cases showed marked conformity with most of the recorded cases of amyotonia congenita: a degenerative condition of the nerve-cells, anterior roots and muscle-fibres without any sign of inflammation and without any lesion of other parts of the spinal cord. Marburg suggests that this condition is due to intra-uterine poliomyelitis. There does not seem to be sufficient reason for accepting this very interesting theory. Marburg supposes that amyotonia is the result of a pre-existing, not an active, process in the spinal cord. It seems, however, impossible to decide if an atrophic pyknoid condition of the nerve-cells is a result of a pre-existing inflammatory process or of a very slow degeneration as in the muscular dystrophies. Marburg is right in assuming that these findings cannot be traced to arrested development. But he cannot point out any certain differences from the progressive muscular dystrophy of the Werdnig-Hoffmann type. To demonstrate the conformity of the recorded cases, both clinically and pathologically, with the progressive muscular dystrophy of the Werdnig-Hoffmann's type, I report the following case.

Case 7.—P. L., born January 27, 1917, son of a job-master, treated at the Children's Department of the Rigshospital, May 30 to June 28, 1918, and March 16 to September 19, 1919. A sister of the patient, aged 5 years, completely well. A father's brother suffered from a congenital paralysis and died at the age of 5 years. Two cousins of the father (his father's sister's sons) had a similar disease; one of them died 16 years old of cerebral affection, the other died 11 months old.

The patient was born naturally, labour lasted only one hour. During pregnancy the mother felt normal fœtal movements at the usual time. He was suckled by his mother for three months, developed normally, smiled and moved himself about freely during the first eight months. At eight months it was remarked that he could not remain erect when raised, later there was no change in this respect. He seemed also from that time to move his legs less well than before; he improved somewhat after treatment with massage. Otherwise he was perfectly well, there were no convulsions or tremors, no squint. The examination (in my private practice) February 6, 1918, when the child was 1 year old, showed the patient to be very fat. Upper extremities, which were hypotonic but not hyperflexible, were moved freely. Lower extremities very flaccid. Tendon reflexes absent. When pricked with a needle he moved his legs slightly. The crural muscles reacted both to the faradic and the galvanic current somewhat slowly, especially on kathode-make.

On admission to the Children's Department of the Rigshospital, May 30, 1918, the child was well nourished. Left-sided cryptorchism; examination of the organs showed nothing abnormal. Urine normal. v. Pirquet reaction negative. Hæmoglobin (Sahli) 87 per cent. Weight 8,620 grm. Fontanelles closed; 4/4 teeth. Stethoscopic examination of the heart and the lungs negative. The child was quick, at one examination only somewhat dull. Nothing abnormal in the cranial nerves.

Trunk.—When he was placed erect, the muscles of the neck and back were flaccid. The thorax was soft and retracted during respiration. Abdominal reflexes present.

Upper extremities were moved freely. The muscles were somewhat flaccid and atonic. *Lower extremities*: The muscles everywhere flaccid and atrophic. He could only draw his legs up a little. Movements of the feet and toes were apparently absolutely free. Tendon reflexes could not be elicited. Plantar reflexes normal.

Electrical reactions: K.C.C. = 5, A.C.C. = 6.15, A.O.C. > 10, K.O.C. > 10.

Later examinations, March 18 and June 22, showed that he could not draw his legs up as at first. He seemed during his stay in hospital to be somewhat backward mentally.

March 16, 1919: The child was again admitted for pneumonia. The mother said that he had recently been rather feeble in spite of treatment with massage. Two days before admission he became febrile and dyspnœic. On admission there were signs of pneumonia on the right side, and the child died in twenty-four hours.

The autopsy showed broncho-pneumonia of both the lungs and purulent bronchitis.

Microscopic examination of spinal cord.—In the lumbar cord the cells of the anterior horns were very markedly diminished in number. A few were quite normal, and a few were very atrophic, pale, thin and angular. In the dorsal and cervical regions the appearance was somewhat different: there were hardly any normal cells to be seen, but a considerable number of abnormal nerve cells. All these cells were diminished in size, angular and slender, but not pale; on the contrary, they were dark-coloured, with pyknoid protoplasm which hid the nucleus. In the dorsal region the cells of Clarke's column were normal in size and shape, but without staining of Nissl's granules.

It should be noted that the difference between my six cases and the progressive muscular dystrophy of Werdnig-Hoffmann's type is very little, both clinically and pathologically. But it must be emphasized that my cases are congenital and so far represent a great rarity, for only very few cases of congenital muscular dystrophy seem to have been recorded. This disease would seem to be much more rare than amyotonia congenita, of which more than sixty cases have been published.

We will, however, make the following comparisons :—

(1) Amyotonia congenita, according to the original descriptions, seems to be a rather benign and not an hereditary disease, and does not seem to leave any defects in adults.

(2) In eight of the eleven cases which have come to autopsy, there have been severe atrophies of the anterior horn cells and muscles.

(3) In two of my cases of hereditary congenital muscular atrophy corresponding changes were seen.

(4) These changes resemble, on the whole, the changes in Werdnig-Hoffmann's progressive muscular atrophy.

(5) In my six cases the disease clinically resembled closely amyotonia congenita, and only later information showed that it was a familial disease.

I would emphasize the radical pathological difference existing between the heredo-familial diseases and other diseases and, remembering that every heredo-familial disease in a certain number of cases is found isolated, I would suggest the following explanation as the most probable :—

The cases which have been described as amyotonia congenita represented really two different diseases :—

(1) One of these, amyotonia congenita (myatonia congenita, Oppenheim), is a benign disease, which consists of a congenital hypotonia, hyperflexibility and weakness, but *no atrophies.* If the patient does not die from intercurrent diseases, it may be assumed that he is cured. It is not familial and can possibly be considered as a retarded development of the muscles.

(2) From this true amyotonia congenita must be separated the following : The cases first described by Beevor[1], Sorgente, Silvestri and Skoog. Secondly the cases in which, at the autopsy, atrophy of the anterior horn cells and muscles was found. They are Rothmann's, Reyer-Helmholtz's, Archangelsky-Abrikosoff's, Marburg's, Collier-Holmes', Griffith-Spiller's, Laignel-Lavastine-Voisin's and Kaumheimer's.

---

[1] Originally considered as myopathy, later as amyotonia.

All these, with Batten's cases, Howard's case, Wimmer's case and Jendrassik's case, and the six cases which I have described, must probably be considered as cases of congenital familial progressive spinal muscular atrophy, a type which is most related to Werdnig-Hoffmann's type, but is congenital. This disease has a certain clinical resemblance to amyotonia congenita, but differs from it in that the muscles show a marked atrophy, demonstrable by the X-rays, and in that it has a tendency to progression or at any rate not to improvement, and in that, for a certain number of cases, it can be shown that the disease is familial.

My best thanks are due to Professor C. E. Bloch for permission to examine cases in his wards and for the interest he has shown in my labours. I also wish to thank Professor Dr. Viggo Christiansen for permission to publish the first case from the neurological polyclinic of the Rigshospital.

REFERENCES

ARCHANGELSKY und ABRIKOSOFF. "Ein Fall von Myatonia congenita Oppenheim mit Autopsie," *Archiv für Kinderheilk.*, 1911, Bd. lvi, S. 101.

BATTEN. "Three Cases of Myopathy, Infantile Type," *Brain*, 1903, vol. xxvi, p. 147.

Idem. "Progressive Spinal Muscular Atrophy of Infants and Young Children," *Brain*, 1910-11, vol. xxxiii, p. 433.

Idem. "Myopathy, Infantile Form of Landouzy-Dejerine's Type," *Proc. Roy. Soc. Med.*, 1914-15, vol. viii, Neurol. Sect., p. 70.

BATTEN and GORDON HOLMES. "Progressive Spinal Muscular Atrophy of Infants" Werdnig-Hoffmann's type, *Brain*, 1912-13, vol. xxxv, p. 38.

BEEVOR. "A Case of Congenital Spinal Muscular Atrophy, &c.," *Brain*, 1902, vol. xxv, p. 85.

CHÉNÉ. "L'atonie musculaire congénitale (maladie d'Oppenheim)," *Thèse de Paris*, 1910, contains 57 references.

COLLIER. "Case of Amyotonia Congenita," *Proc. Roy. Soc. Med*, 1914-15, vol. viii, Neurol. Sect., p. 71.

COLLIER and HOLMES. "The Pathological Examination of Two Cases of Amyotonia Congenita, with the clinical description of a fresh case," *Brain*, 1909, vol. xxxii, p. 187.

COLLIER and WILSON. "Amyotonia Congenita," *Brain*, 1908, vol. xxxi, p. 1.

CONCETTI. "Ancora sulla atonia muscolare congenita di Oppenheim, in specie dal punto di vista anatomo-pathologico," *Riv. di clin. pediatr.*, 1913, t. xi, p. 1.

FEARNSIDES, E. G. "Case of Amyotonia Congenita (? Atonic Form of Cerebral Diplegia)," *Proc. Roy. Soc. Med*, 1914-15, vol. viii, Neurol. Sect., p. 49.

FOOT. *Amer. Journ. Diseases of Children*, 1913, vol. v, p. 359.

GRIFFITH and SPILLER. "Amyotonia Congenita: a Clinical and Pathological View," *Amer. Journ. of Med. Sciences*, 1911, vol. cxlii, p. 165.

GUTHRIE. *Trans. Clinical Society*, 1899, vol. xxxii, pp. 262, 263.

JENDRASSIK. *Deut. Zeitschr. für Nervenheilk.*, 1902, Bd. xxii, S. 444.

Idem. In Lewandowsky's "Handbuch der Neurologie," Bd. ii.

KAUMHEIMER. "Zur Pathologie und Klinik der Myatonia congenita" (Oppenheim), *Jahrbuch fur Kinderheilk.*, 1913, Bd. lxxviii, Suppl., S. 170.

LAIGNEL-LAVASTINE et VOISIN. "Note histologique sur la myatonie congénitale," *Revue neurologique*, 1913, t. xxvi, p. 46.

MARBURG. "Zur Klinik und Pathologie der Myatonia congenita" (Oppenheim), *Arbeiten aus dem Neurol. Inst. Wien.*, 1912, Bd. xix, S. 133.

REUSS, A. "Die Krankheiten der Neugeborenen," Berlin, 1914.

SKOOG. "Myatonia congenita," *Zeitschr. f. d. ges. Neurol. u. Psychiat.*, 1914, Original, Bd. xxvii, S. 357.

SPILLER. "The Relation of the Myopathies," *Brain*, 1913-14, vol. xxxvi, p. 75.

THORSPECKEN, "Beitr. zur Kenntnis der Myatonia congenita" (Oppenheim), *Jahrbuch fur Kinderheilk.*, 1912, Bd. lxxvi, S. 300.

WHAIT. "A Case of Amyotonia Congenita," *Proc. Roy. Soc. Med*, 1907-8, vol. i, Neurol. Sect., p. 61.

WIMMER. "To Tilfælde af kongenit Muskellidelse hos Born," *Nordisk Tidsskrift for Terapi*, 1905-6, Bd. iv, S. 193.

Idem. "Zwei Fälle von congenitalem Muskelleiden bei Kindern," *Archiv fur Psychiatrie*, 1907, Bd. xlii, S. 960.

ZIEHEN. "Beziehungen zwischen angeborenen Muskeldefekten, infantilem Kernschwund und Dystrophia musculoris progressiva infantilis," *Berl. klin. Wochenschr.*, 1908, Bd. xlv, S. 1557.

PUBLICATIONS RECENTLY RECEIVED.

[*Notes on a Book under this heading do not preclude a subsequent review.*]

The Conduction of the Nervous Impulse. By KEITH LUCAS, SC.D., F.R.S. Revised by E. D. ADRIAN, M.D., M.R.C.P. Pp. 102. London: Longmans, 1917.

This remarkable work made its appearance during one of the most difficult periods of the war, and failed to attract the notice it deserved. Dr. Keith Lucas delivered a course of seven lectures at University College, London, in the spring of 1914, and intended to publish them as a monograph. At the outbreak of War he joined the Army and was posted to the Royal Aircraft Factory. There he was killed in an aeroplane accident on October 5, 1916. After his lamentable death, Dr. Adrian completed the manuscript, and wrote several of the chapters either from notes left by Dr. Lucas, or from his knowledge of the views held by his fellow-worker and friend. Most practical neurologists are not acquainted with the revolution created by these researches in our knowledge of the nature and conduction of the nerve impulse. This little book is therefore greatly to be welcomed, because the views of Keith Lucas are here put together in an accessible form, and expressed in the simplest possible language. It is worthy of close study by all who are interested in the modern views of the physiology of the peripheral nervous system.

Travaux neurologiques de Guerre. By GEORGES GUILLAIN and J. A. BARRÉ. Preface by Professor PIERRE MARIE. Pp. 463. Paris: Masson, 1920.

In this volume the authors have collected together their valuable contributions to the literature on the effects of injuries of the nervous system in war. They cover a vast field, for sections are devoted to wounds of the head, of the spinal cord and of peripheral nerves, the study of reflexes in the lower extremities by graphic methods, and a variety of other subjects. Much of their work was carried out close to the firing line and is of especial interest, since it deals with the early effects of acute lesions of the nervous system. For example, they were able to study the results of transection of the spinal cord in fifteen patients almost immediately after they were wounded, and their description of the findings is unique. The chapter on Concussion of the Nervous System is particularly fascinating. No less than 789 patients with injuries of this class passed through their hands, and many rare conditions, such as epileptic crises, generalized contracture and disorders of co-ordination resembling those of disseminated sclerosis, were observed during the early stages of the illness.

The book is a record of investigations carried out with extreme care and under great difficulties by skilled observers and fully merits the laudatory remarks of Professor Pierre Marie in his preface.

Mammalian Physiology, a Course of Practical Exercises. By C. S. SHERRINGTON, M.D., D.Sc., F.R.S. Pp. 156, with many illustrations. Oxford: Clarendon Press, 1919.

This remarkable book purposes to set out a series of practical exercises for a class in physiology; but the novel feature is the extensive use made of the decapitate or decerebrate mammalian preparation. This takes the place of the frog in most of the experiments, and the student is able to make his observations on a warm-blooded animal, which has been pithed at a higher or lower anatomical level. "The actual performance by the student," says the author, "of some few such main experiments gives him, I am convinced, a better insight into their general significance and into the problems they touch, than does any mere inspection at the demonstration, however skilfully conducted." All the graphic records are those made by students themselves, and in many cases their names are immortalized at the foot of the curve. Many of us can only look with envy on the happy members of such a class.

Mikroskopischer Atlas des menschlichen Gehirns. Herausgegeben von Professor Dr. G. FUSE und Professor Dr. P. C. von MONAKOW. I. Die Medulla Oblongata. Consisting of plates, 44 × 59 cm. Zurich: Orell Füssli, 1916.

This atlas contains six large plates representing transverse sections of the medulla oblongata. They were drawn by Professor G. Fuse, of Japan, in the Institute of Professor von Monakow. On each plate the parts are named so that an explanatory text is unnecessary. The various structures have as far as possible been checked by secondary degenerations and comparison with other collections of similar microscopical preparations. If the subsequent portions of this work approach in accuracy and fulness the present atlas, it will be a most valuable guide to future workers in organic pathology of the nervous system.

Les Médications psychologiques. I. L'Action morale, l'Utilisation de l'Automatisme. Pp. 343. II. Les Économies psychologiques. Pp. 307. III. Les Acquisitions psychologiques. Pp. 494. Three volumes paged independently with a general index at the end of Vol. III. By Dr. PIERRE JANET, Professeur de Psychologie au Collège de France. Paris: Félix Alcan. 1919.

In these three volumes, Professor Pierre Janet has attempted to put together the nature and action of the various means that have been used at different periods in the world's history for combating the psychoneuroses. He first deals with "Moral Action." This he divides into miraculous cures under the influence of religion, magic or animal magnetism; then he takes philosophic treatment such as Christian Science, and finally what he calls "Moralisation médicale," of which he gives as his example treatment by Dr. Dubois of Berne.

The second part of the first volume is devoted to the forms in which automatic action is used to produce a cure. Here, he deals with the whole history of hypnotism and suggestion. This is a most valuable summary of the forms employed at various times and the aims of the different schools of thought.

The second volume is devoted to treatment by rest, by isolation, and by what Janet calls "Liquidation morale." As this comprises psycho-analysis and Freud's views of the nature of these disorders, it will be obvious that the term "psychological economy" has been made to cover an unusually wide ground.

The third volume is devoted to what the author calls "Acquisitions psychologiques" such as re-education, the production of crises and somnabulisms, the treatment by excitation and moral direction.

It is obviously impossible to give any idea of the contents of this work in a short space. It is a book that must be studied carefully, and is full of information with regard to the various forms of treatment that have been adopted from time to time. It is, however, doubtful whether the author was wise to divide the various sections in so logical a manner; for many of the views that have been held at different periods do not fall into their proper position, unless they are treated in sequence; for example, it is important to remember the part played by Janet's own work in the evolution of Freud's views, and subsequently those held by Jung and others.

The third volume is a valuable index of authors cited and of the subjects treated throughout the work.

Functional Nerve Disease. Edited by H. CRICHTON MILLER, M.A., M.D. Pp. 208. London: Henry Frowde, Hodder and Stoughton, 1920.

Dr. Crichton Miller, as general editor, has gathered together short contributions from ten authors on the subject of functional nervous disorders. The book is divided into four main portions; these deal with the physical, hysterical and anxiety factor, followed by two chapters on the management of the neurotic, and a summary by Dr. McDougal. The various chapters are in reality independent of one another, for each author is permitted to hold his own views, and does not hesitate to criticize the different schools to which his colleagues belong. For those readers who are already familiar with the present trend of psychopathology, it is of great interest to have the various views presented with such clearness. Each chapter is prefaced by a synopsis which enables the reader to appreciate the tendency of the ideas put forward by the writer; were this not the case, the concentrated writing of some of the chapters would be somewhat difficult to understand.

Treatment of the Neuroses. By ERNEST JONES, M.D. Pp. 233. London: Baillière, Tindall and Cox, 1920.

This is an interesting little book written with all the ease and clearness we have grown accustomed to expect of this author. Although it is supposed to be concerned with the treatment of the neuroses, the main portion of the book is occupied with hysteria, and the remaining functional disorders are treated comparatively shortly. This is, however, justified by the fact that when discussing hysteria and the various views that have been held as to its nature, Dr. Jones deals with most of the processes which underlie the general neuroses. To whatever school the reader may belong, this book can be strongly recommended as an introduction to modern views on this subject.

Psychoneuroses of War and Peace. By MILLAIS CULPIN, M.D., F.R.C.S. Pp. 127. Cambridge: University Press, 1920.

Every writer on the psychoneuroses who has had any extensive personal experience, must of necessity adopt an individual attitude towards these disorders. Dr. Culpin worked originally as a surgeon, and was struck with the large number of cases in which psycho-pathological states were mistaken for surgical or medical conditions. Finally, towards the end of the war, he obtained an opportunity of working under more favourable conditions in an institution devoted to the neuro-psychoses; this monograph is the result of his final experience It is somewhat loosely written, but is full of interesting material. The way in which the cases are strung together in the text makes it somewhat difficult to read for the practitioner who is not familiar with the conditions described. But it is of great interest to those who have followed the recent trend towards the recognition of the psychical element which forms so important a part in what appear to be straightforward surgical or medical cases.

A Manual of Neurasthenia. By IVO GEIKIE COBB, M.D. Pp. 366. London: Baillière, Tindall and Cox, 1920.

This is a systematic attempt to construct clinical signs and symptoms which accompany what is called neurasthenia in its widest sense. The mental aspects are treated shortly, and then various symptoms such as headache, gastro-intestinal disturbance, sensory disorders, insomnia, and what are called "objective signs" of neurasthenia are dealt with seriatim. The second part of the book deals with treatment, consisting of general hygiene, diet, the use of drugs, climate and electricity: psycho-therapeutic treatment occupies thirteen pages only.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[NOVEMBER, 1920.]

CALCUTTA UNIVERSITY
Acc. No.........
Book No.........
LIBRARY.

BRAIN.

PART 3, VOL. 43.

ON THE SYMPTOM-COMPLEXES OF LETHARGIC ENCEPHALITIS WITH SPECIAL REFERENCE TO INVOLUNTARY MUSCULAR CONTRACTIONS.

BY F. M. R. WALSHE, M.D., B.Sc., F.R.C.P.
Medical Unit, University College Hospital, London.

PAGE
I.—INTRODUCTION 197
II.—CLINICAL CASES 202
III.—INVOLUNTARY MOVEMENTS AND MUSCULAR CONTRACTIONS: THE SO-CALLED "MYOCLONIC" AND "CHOREIFORM" MANIFESTATIONS OF LETHARGIC ENCEPHALITIS CONSIDERED AS IRRITATIVE PHENOMENA. THE CONTRAST BETWEEN THE PARALYTIC AND THE EXCITING ACTION OF THE VIRUS 213
IV.—SUMMARY AND CONCLUSIONS 218
REFERENCES 219

I.—INTRODUCTION.

WHEN a disease, particularly one involving the central nervous system, is isolated in the first place by clinical observation, it follows of necessity that only cases corresponding to a single symptom-complex can be identified as belonging to it. With increasing knowledge the originally clear-out conception of the malady becomes inadequate to include the inevitable variations from type that are observed. It has to be broadened and it loses the artificial precision of its earliest form.

Investigations into the ætiology and morbid anatomy of the malady proceed simultaneously with the extension of knowledge on the clinical side, and, finally, it becomes possible with reasonable accuracy to describe a clinical picture characteristic of the disease and to establish its main clinical variations and forms. It may be possible in some cases to correlate these with variations in the intensity or distribution of the underlying lesions in the nervous system.

It is clear that during this phase of growing experience much modification and revision of opinion as to the significance and value of observed phenomena may be necessary.

In the case of lethargic encephalitis we are still in this phase of growing knowledge. We know little of its ætiology and the full range of its clinical manifestations has probably not yet been determined. In the absence of any bacteriological or serological standards it is not always possible to decide whether a given symptom-complex should be included in the rapidly growing group attributed to this disease or not. It seems probable from a study of the literature that from time to time cases are incorrectly included, while others that belong to the group may equally well be rejected from lack of these standards.

The original clinical picture by which, under the name of botulism, the disease was first diagnosed in this country, soon gave place to various forms or types, the number of which is steadily on the increase. As Buzzard and Greenfield [5] have pointed out, it is to be expected that an inflammatory disease of the nervous system should give rise to a versatile symptomatology; the structure of this system is so complex and the functions of its several parts so highly specialized. Indeed, it is easy to conceive of innumerable possible combinations of symptoms—that is of possible syndromes—we might reasonably expect to encounter, though it would not be correct to call them all "types."

Many schemes of classification for the various syndromes of lethargic encephalitis are to be found in the literature. Most of them are purely symptomatic; some prominent symptom in a series of cases being chosen as the basis for the definition of a type. Other writers employ mixed anatomical and symptomatic groupings. The result has been that each case either simultaneously or at some stage of its course falls into almost as many groups as it has symptoms. The recently named "myoclonic encephalitis" affords a good example of this. Rhythmical clonic contractions and fibrillary twitchings of muscles, or of parts of muscles, have been recorded in lethargic encephalitis by numerous observers during the past two years. In the recrudescence of the disease during the past winter (1919-20) they have been a remarkably prominent feature. In some recently reported cases they have dominated the clinical picture entirely (Ellis, [10]), while in a few instances (Sicard and Kudelski [17, 18, 19]) clonic muscular contractions have been the only somatic nervous symptom observed. At first sight it might seem justifiable, with Sicard, to regard so clear-cut and striking a symptom-complex as a new and distinct type of the disease. However, experience indicates that the particular combination of symptoms recorded by Sicard is extremely rare in a pure form; that usually it occurs either superimposed upon, preceding, or following a whole series of other symptoms in any given case; and that clonic muscular contractions occur as a symptom in cases of every clinical variety. Nevertheless, following Sicard, numerous writers now group as "myoclonic encephalitis" cases differing in the widest possible manner and often having nothing in common clinically except clonic twitchings of one or more muscles or parts of muscles. Moreover, the term "myoclonic" has been applied to such a variety of involuntary muscular contractions and movements by French writers that it has come to have little or no meaning. The unsatisfactory nature of classification of this kind is exemplified by such a case as that reported by Bourges and Marcandier [4] as a "forme mixte à symptômes intriqués (co-existence de somnolence, paralysies partielles oculaires, mouvements choréo-athétosique, secousses myocloniques, catatonie et troubles délirants"). While Kahn reports [12], "Un cas d'encéphalite successivement névralgique, psychique, choréiforme, myoclonique, léthargique avec séquelles hémi-myoclonique après la guérison."

Such complex syndromes are by no means exceptional and the disease which can produce them clearly defies symptomatic classification.

Quite possibly no permanent classification can be devised in the present incomplete state of knowledge, but it is reasonable to suppose that an analysis of the features of the disease might reveal some fundamental character upon which a uniform scheme of description could be founded.

The symptoms of lethargic encephalitis fall naturally into the following groups:—

GENERAL SYMPTOMS—Signs of toxic infection ..			Fever. Shivering. Cutaneous eruptions. Pains and malaise. Headache. Gastro-intestinal disorders, such as vomiting, constipation, foul tongue, &c.
NERVOUS SYMPTOMS	General	"Positive"	Delirium, mania, restlessness, hallucinations, &c.
		"Negative" ..	Lethargy, coma.
	Focal ..	"Positive"	Convulsions. Involuntary movements and muscular contractions. Ataxy, rigidity. Pains.
		"Negative" ..	Paralyses.

CALCUTTA UNIVERSITY
Acc. No.
Book No.

The term "positive" and "negative" are employed in the sense in which Hughlings Jackson originally used them—"positive" symptoms being expressions of exalted function either from irritation or from loss of higher control, and "negative" symptoms expressing depression or loss of function from destruction of nervous structures or otherwise. A recognition of these two classes of symptoms is essential to a comprehension of any nervous disease.

Not all these symptoms are represented in a single case, nor are those present of equal value in every instance. Thus, the general symptoms of toxic infection are minimal or entirely escape observation in some instances, while in others they dominate the clinical picture at the onset or even throughout the illness.

If ten consecutive cases were taken and considered in the light of the scheme given above, it is quite possible that not a single symptom would be common to them all. If we seek for a common factor we shall find it only in the presence of focal or localizing nervous symptoms.

Among those symptoms that are more definitely focal in origin, the localization of the positive motor phenomena presents considerable difficulty, and very diverse views have been expressed as to their cause and site of origin. Therefore, as they may be the only focal symptoms present in a given case, an attempt to analyse their nature and cause is an essential preliminary to any sound basis of description or classification. This question will be considered later, but in the meantime a few general observations on the symptomatology of lethargic encephalitis may be made.

At the time of its first recognition, the disease was remarkably "negative" in the character of its symptoms: lethargy and paralyses of certain motor cranial nerves made up its clinical picture in most instances, and although certain "positive" general and focal nervous manifestations were recorded from 1918 onwards, they were certainly not so characteristic of the malady as they have since become. Doubtless, there are many factors responsible for this apparent change in the clinical aspect of lethargic encephalitis during the past few months. It is very easy to exaggerate the importance of this change. Undoubtedly a widened conception of the disease and the admission of cases that could not have been recognized earlier as belonging to it have been contributory causes. But it also must be remembered that although overlaid by more obtrusive positive symptoms, the same negative symptoms that attracted attention to the disease originally still occur with considerable frequency. As Netter [15] has pointed out in this connection, it has long been known that a poison may cause either exaltation or depression of function, at one time producing convulsions, at another paralysis, as alcohol, lead, and the virus of rabies are well known to do. Further, the clinical variations of an infective disease in successive epidemics are quite familiar, and although there certainly is some evidence of more extensive involvement of the nervous system now than in the earlier cases, yet we need not regard a change in the localization of the virus as even the most important of the many factors concerned.

Whatever their cause may be, the frequency and variety of "positive" symptoms, both general and focal, have increased the clinical diversity of lethargic encephalitis still further, so that a comprehensive view of its essential features is becoming increasingly difficult.

Nevertheless, an appreciation of the fact that the same principles govern the localizing of "positive" as of "negative" symptoms, of a clonic contraction as of a palsy, renders the problem less complex than appears at first sight and makes the prospect of finding a satisfactory uniform basis of description more hopeful.

A consideration of *acute poliomyelitis* in the light of the scheme given above offers many interesting points of contrast between that disease and lethargic encephalitis. In the former the general symptoms are far more constant in form and duration than in lethargic encephalitis. They always precede the onset of paralysis and rarely last more than four or five days. When they abate the activity of the virus may be considered to be spent. In lethargic encephalitis, on the other hand, as we have seen, they vary greatly in intensity, form and duration, and, as Netter [15] has suggested, it is probable that the virus remains active for weeks or months. The nervous symptomatology of poliomyelitis is characteristically "negative" and predominantly focal in type. Yet even in this disease, when the cerebrum is involved, convulsions may occur. In this connection a case described by Leonard Parsons and quoted by Batten [3] as one of poliomyelitis is of considerable interest at the moment. It was that of a child of 2, who was suddenly taken ill with fever, a fit and sleepiness. The fits recurred next day; the child regained consciousness but remained for three weeks "apparently conscious, took her milk, but her eyes were fixed and staring, and she did not appear to see anything nor did she speak." For three weeks her limbs showed a slow rhythmic tremor and were rigid. There was no paresis nor any change in the reflexes. Recovery ensued after five weeks. In 1913 this case was diagnosed as poliomyelitis, it is doubtful if it would be so called now. With rare and questionable exceptions such as this, it may be said that the nervous symptoms of poliomyelitis are negative, or paralytic, and do not show the selective and restricted character of the palsies seen in lethargic encephalitis, which commonly involve certain motor cranial nerves only. For the long-continued fibrillary and clonic contractions of muscles seen in the latter disease there is no parallel in poliomyelitis.

In poliomyelitis classification and description have an anatomical basis. The disease is much less polymorphic than lethargic encephalitis, and its focal symptoms are readily interpreted. Yet even here Wickmann found it necessary to add an "abortive" group of cases to his purely anatomical grouping. There is no reason why in the case of lethargic encephalitis a similar uniform basis of description should not be attempted, though, undoubtedly, the problem is far more complex. Nevertheless, nothing but good can come from the abandonment of the numerous symptomatic classifications that serve only to obscure our outlook on the disease.

II.—CLINICAL CASES.

In this section are briefly described seven cases which were observed successively in the practice of University College Hospital. Five of them were under the care of the Medical unit, and for permission to record the remaining two I am indebted to the courtesy of Sir John Rose Bradford (Case 1) and Dr. F. J. Poynton (Case 5).

These cases were not selected, and their clinical diversity is not, therefore, artificial, but represents the disease as it has been observed in the practice of a single hospital during the past few months, though the cases are not recorded in the order in which they were seen.

At the head of each case the symptoms are summarized according to the scheme given in the first section of this paper.

Case 1.—*Lethargy and Signs of a Cortical Lesion.*

General Symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul tongue, congested fauces, headache and diffuse pains

Nervous Symptoms. — (General) Lethargy passing into coma, general muscular hypotonus and diminished tendon reflexes. (Focal) Right hemiparesis and Jacksonian fits.

Alice R., married, aged 42. On January 12, 1920, she suddenly came over faint and giddy while at housework. She lay on her bed and went to sleep. During the following week she complained of headache, lassitude, vague, fleeting pains in limbs and body, and increasing drowsiness, so that at times she could hardly keep awake. She was very breathless on exertion. For about a week she kept at her work, and her husband appeared to notice nothing abnormal in her. She had previously enjoyed excellent health.

On January 19, 1920, she suddenly became very much worse; she noticed that she was weak on the right side, and that her right arm and leg were twitching.

On examination on the following day she was very somnolent and slept unless aroused This was easily done, however, and she could then give a rational account of her illness Temperature was 103° F., pulse 132, and respirations 32. Her tongue and lips were dry and coated and the fauces congested The thoracic and abdominal viscera appeared healthy. The urine was normal. She complained of headache and of pain in the right side of the chest.

At increasingly frequent intervals, until her death on February 9, 1920, there were right-sided Jacksonian fits of face, arm, hand and leg. The clonic jerkings began in the thumb and index-finger and spread up the arm. The lower part of the face on that side and the leg were simultaneously involved. The movements, which occurred at the rate of about three in two seconds, were most forceful and enduring in the hand and forearm; the leg was least affected. In the upper limb the flexor group seemed most affected; in the leg only the limb extensors and plantar flexors could be felt to contract. Associated with this condition was a progressive right hemiparesis, in order of severity, hand, arm, face, leg. The muscles were flaccid. All tendon jerks were sluggish, right and left equally. No abdominal reflexes were obtained, and the right plantar response was extensor, the left normal. As far as could be determined there was no disturbance of cutaneous sensibility; other forms could not be tested. There was incontinence of urine and fæces.

The patient gradually became comatose, broncho-pneumonia developed, and she died four weeks from the onset of the illness. At no time were there delirium, pain, or ocular paralyses. There was no increase of lymphocytes in the cerebrospinal fluid.

At post-mortem the soft meninges appeared congested, but no other abnormality was seen. The precentral and post-central gyri and the basal ganglia of both sides, the cerebellum, midbrain, and medulla were examined microscopically. In the cerebral cortex and the basal ganglia there was considerable round-celled infiltration of the perivascular spaces, but no other lesion was noted. No difference could be detected between the two sides. The cerebellum and brain-stem were normal.

In this case the focal symptoms indicated a combined irritative and destructive lesion of the cerebral cortex; the paresis and the convulsions having the same localizing value.

Case 2.—Lethargy and Signs of Basal Ganglia and Tegmental Lesions.

General symptoms.—Constipation, fleeting limb pains.

Nervous symptoms.—(General) Lethargy. (Focal) Defective upward movement of eyes, nystagmus, mask-like face, rigidity and tremor of left limbs.

Harry T., aged 14. His mother stated that five weeks before his admission to hospital he came home from work on a Saturday afternoon complaining of being very sleepy. He lay on his bed and slept with intervals, when he was waked for meals, until Monday morning, when his drowsiness was still unabated. During the succeeding five weeks he went to sleep whenever he was left to himself, but was always easy to rouse. He ate his meals fairly well and seemed "right in his mind," though his mother noticed that he "grinned silly" at her when spoken to, and that his left arm and hand shook and were clumsy when in use. He was very constipated, and complained from time to time of vague pains in his limbs. These were never severe.

On examination (July 5, 1920) he lay in bed apparently asleep. He awoke and became quite alert when addressed and could answer simple questions readily and accurately. He displayed a complete loss of initiative, and neither moved nor spoke unless made to do so. His face was vacant and expressionless, but periodically a slow smile dawned on his face, spread, and remained fixed often for as long as a minute before it faded slowly away. Articulation was normal, and he could swallow normally. Vision and hearing normal.

The pupils were normal. Upward movement of the eyes was distinctly limited in range. Lateral movements were normal in range and association. On fixation, particularly on lateral deviation, a coarse, rapid nystagmus appeared. This was better sustained and more rapid on looking to the left than on looking to the right. No vertical nystagmus, no squint or diplopia. No ptosis.

There was moderate paresis of both halves of the face, especially of the upper part; thus the frontales were extremely weak. In movements carried out to order the right half of the face acted more rapidly and amply, but on smiling the left half " took up " much sooner and acted better than the right. The other cranial nerves showed no defect.

Motor system: The left arm and hand were held flexed and abducted, but otherwise his general attitude was in no way peculiar. All mass movements were carried out very slowly as though he were stiff or sore. He moved and walked just like a case of paralysis agitans, and retropulsion was easily induced by making him hold back his head or by a slight push. The left arm and leg showed a curious "sticky" rigidity like that seen in paralysis agitans, and clearly differing from that seen in lesions of the pyramidal system. Movements of the left hand and digits were very slow and clumsy and a fine tremor of the forearm and fingers appeared on movement. The leg also was slow in movement but there was no tremor. Isolated movements of the right limbs were carried out with normal facility and speed, but in mass movements the pace seemed to be set by the capacity of the affected left limbs.

There was no weakness of the left limbs, power and muscular development were normal throughout. The tendon jerks were of normal facility and equal on the two sides. The abdominal reflexes were all present and the plantar reflexes flexor in type. Sensation was normal and there was no pain. Sphincter control was normal.

There was no fever. The cerebrospinal fluid was normal in every respect, there was no lymphocytosis or increase of globulin. Wassermann negative.

During the following four weeks the condition altered very little, but the lethargy gradually lessened.

At present (September 1, 1920) there is no lethargy, the rigidity and slowness of movement of the left arm are improved, but general movements, the defect of eye movements, the facies and nystagmus are unaltered.

In this case the focal symptoms indicate a lesion of the basal ganglia and the tegmental region of the midbrain. The unilateral character of certain of the symptoms is unusual, but in other respects they are of a kind quite familiar and may be regarded as examples of a mechanism released by defective activity of co-operative centres.

Case 3.—Delirium and Extreme Restlessness (Choreiform) followed by Lethargy. Signs of Midbrain (peri-aqueductal) Lesion.

General symptoms.—Fever, breathlessness on exertion, anorexia, constipation, foul dry tongue and lips, morbilliform eruption and joint pains.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations, extreme restlessness resembling severe chorea. All passing suddenly into profound lethargy after thirty-six hours. General muscular hypotonus and diminished tendon jerks. (Focal) Third, fourth, sixth and seventh nerve palsies.

Louisa F., aged 14. On May 24, 1920, the patient's mother noticed that she was very short of breath on exertion. Two nights later she began to be sleepless and restless at night. This became nightly worse, so that on the night before admission she was violent, could scarcely be kept in bed, threw herself about, appeared to be having terrifying visions and shouted that men were after her. At the same time "a rash suddenly came out on her like measles." During this period of onset she was constipated, had no appetite, complained of severe pain in the right wrist which made her cry out at times, and she seemed feverish. Admitted to hospital as a case of chorea.

On admission (May 31, 1920): She lies on her back in bed, continuously tossing about. She flings her head and limbs about with great force and has to be kept from falling out of bed by a strong sheet. The condition resembles severe adult chorea. She is flushed, her skin is hot and dry, her lips are dry and cracked and her tongue coated. Temperature 102° F.

Over legs, elbows, buttocks and shoulders there is a bright red papular rash. At the apex of the papules are small dark red crusts like dried hæmorrhages.

Although this is most marked over areas where friction is greatest it is also present on protected areas of skin and cannot be entirely due to rubbing.

Nervous system: She is in a muttering stupor when not disturbed, but when spoken to half opens her eyes and gives rational replies to simple questions, though she can give no account of her illness. Her answers are perfectly articulated.

Vision and hearing appear unaffected. She does not complain of tinnitus, but says that she sees double.

Cranial nerves.—Her pupils are equal, central and circular. They react fairly well to both light and accommodation. There is marked double ptosis with feeble overaction of the frontales when she opens her eyes. The eyes do not move at all parallel, but no single muscle can be incriminated and all movements appear limited in range. There is no nystagmus. There is diplopia. There is profound weakness of both sides of the face. She can close her eyes, but there is scarcely any movement round the mouth. Her tongue is protruded straight and well and can be maintained steadily protruded. All its movements are perfectly normal. The motor and sensory fifth nerves are normal. Articulation, deglutition and phonation are normal.

Motor system.—There is general hypotonus and some general weakness, but no true paresis, nor localized weakness. The constant movements of head, trunk and limbs closely resemble those of chorea, but there appears some evidence of voluntary control when carrying out movements to order. The steadiness of the tongue moreover is unusual in severe chorea. These two facts and the general character of the movements suggest that although the resemblance to chorea is striking it is mainly superficial and the presence of the cranial nerve palsies confirms this view. The tendon jerks are all sluggish, but obtainable. They are equal on the two sides. Abdominal reflexes doubtful. Plantars flexor. There is no demonstrable defect of sensation. She complains of pain in the region of the right wrist, but no local abnormality of any kind can be detected.

(June 2, 1920.) After eighteen hours the movements ceased suddenly and the patient subsided into a profound lethargy. She now resembles the ordinary form in which lethargy and signs of a peri-aqueductal lesion co-exist. Temperature remains raised. She can be aroused for feeding and to answer questions. The pain in the right wrist has gone.

June 15, 1920. During the past two weeks the cranial nerves have rapidly regained normal function. There is now very slight ptosis. The eye movements are normal and there is no facial weakness. She is still somewhat lethargic and sleeps most of the day. At night she is more wakeful and complains of pain in the region of the left shoulder. This pain is localized in distribution and not neuralgic in character. There is no local condition to account for it. The rash has disappeared and the general condition is good.

On admission the cerebrospinal fluid was examined. There were 160 lymphocytes to the cubic millimetre. There was no leucocytosis in the blood.

The extreme restlessness accompanying the delirium when the patient first came under observation closely resembled severe chorea, and it was with this diagnosis that she was admitted to hospital. The presence of cranial nerve palsies, the sudden onset, and certain minor features which are emphasized in the foregoing account sufficed for the recognition of lethargic encephalitis.

The extreme violence of the movements, their incessant and disorderly character and their association with marked psychical excitement make it extremely probable that we are dealing here with an irritative lesion of higher cerebral centres, rather than any release mechanism.

Case 4.—Lethargy and Acute Cerebellar Ataxy

General symptoms.—None observed.

Nervous symptoms.—(General) Acute onset of headache, vertigo and vomiting. (Focal) Acute cerebellar ataxy.

Captain F. C., aged 27. On May 5, 1920, after a hearty breakfast, and having been previously in apparently excellent health, he was suddenly seized with severe headache, giddiness and vomiting. These symptoms persisted throughout the day. He was examined during that evening and then lay curled up on his bed on his right side. He showed extreme photophobia, but was not irritable. He was fully conscious and rational, but could not give a good account of his symptoms on account of a gross defect of articulation. His friends stated that he had "stammered badly" all that day, though he had never been known to do so before. There was no fever, pulse rate was 64, and respiration rate 20.

Re-examined in hospital on May 7, 1920. His general condition was improved, the vomiting had ceased and the headache diminished, though some occipital pain remained and was aggravated by movement. There was no sense of giddiness. He was distinctly lethargic and lay curled up in bed and apparently asleep until spoken to, when he became quite alert and rational.

There was extreme ataxy of articulation, which was slow and accompanied by overaction of the facial muscles.

Vision: acuity, fields and fundi, normal. Hearing normal, some tinnitus. The pupils were equal and reacted normally. There was no ptosis, or diplopia, but there was a slight divergent squint of the right eye, probably congenital. On lateral deviation a fine rapid nystagmus appeared.

There was a doubtful paresis of the lower part of the left side of the face. Deglutition and phonation were normal. The tongue was protruded normally.

There was gross ataxy of the arms of cerebellar type, the right arm was particularly unsteady and jerky on movement. He walked with a wide base and had to be supported. There was no tendency to fall in any constant direction. The tendon reflexes were brisk and equal on both sides, the abdominal reflexes were present. The left plantar reflex was doubtfully extensor in type. There was no discoverable defect of sensation.

The cerebrospinal fluid was normal in cell and globulin content. The Wassermann reaction was negative. There was no leucocytosis in the blood.

During the six weeks after admission all these symptoms gradually cleared up and on discharge in June slight ataxy of speech, which was still somewhat staccato in character, and some intention tremor of the right hand and arm, were the only noticeable symptoms. When fatigued some unsteadiness of gait appeared.

In this case the focal symptoms are those known to be associated with a negative or defective lesion of the cerebellum. They are in no way peculiar to lethargic encephalitis.

Case 5.—Lethargy and Signs of Irritation of Lower Motor Neurones.

General symptoms.—Lassitude, anorexia and constipation.

Nervous symptoms.—(General) Insomnia, nocturnal delirium, hallucinations passing into somnolence. General muscular hypotonus and diminished tendon jerks. (Focal) Rhythmic contractions of certain arm and abdominal muscles.

George C., aged 44. (History obtained from friends.) Had a "chill" last April. Since this he has not been well. He has complained of being tired, without appetite, and constipated. On May 26 he began to be sleepless at night and to mutter in his sleep. During the following week he complained of severe pains in his limbs, was told by his doctor that he had rheumatism and advised to stop work. Early in June he had severe pain in the chest and was thought to have pleurisy. Since the first week in June he has been confined to bed, semi-unconscious during the day and restless and violent at night. He appeared to be having terrifying visions, talked excitedly and had to be kept in bed by force. He was incontinent of urine and very constipated.

On admission to hospital (June 19, 1920) he was profoundly lethargic, but could just be roused to answer simple questions. He could give no account of his illness. He understood spoken speech perfectly and replied in whispers. He complained of no pain. He passed urine in the bed. Temperature was 101° F., pulse 128, and respirations 32.

Hearing and vision appeared normal. Fundi were normal. The cranial nerves showed no defect. There was general muscular weakness and hypotonus, but no localized paresis. There was no spontaneous tremor or muscular contraction. No sensory loss could be detected. The tendon jerks were all very sluggish, the abdominal reflexes not obtainable and the plantars flexor.

The cerebrospinal fluid was normal in every respect, and there was no leucocytosis in the blood.

During the following two months his general condition and his lethargy remained unchanged and he became slowly thinner. It was found during this period that on passive manipulation of the arms, and after such forceful voluntary contractions of the muscles of these limbs as he could be induced to carry out, there appeared slow clonic contractions of certain forearm and small hand muscles. The rate of these was fairly constant and was about 48 per minute. These contractions which involved the whole muscle appeared first in the supinator longus, then in the extensor group of muscles, and finally in the thenar muscles. They were feeble, and after two or three minutes they died away until the limb was further manipulated.

Similarly, abdominal palpation evoked feeble rhythmic contractions of the upper segments of the recti on both sides, which caused the umbilicus to be drawn upwards at each twitch. Very occasionally the right sternomastoid was seen to twitch spontaneously in the same manner and at the same rate.

September 1, 1920. The patient's general condition is gradually failing, he is losing weight and a large sacral bedsore has appeared. The lethargy remains unchanged and he can still be aroused and can carry out simple orders and answer simple questions in a whisper.

The rhythmic contractions described above have undergone an interesting change. Passive stretching of the forearm and hand muscles now evokes fibrillation and occasionally fascicular twitching of large strands of muscle. If passive manipulations be maintained for several seconds a slow rhythmic contraction, very feeble and producing very little movement of the digits, of the flexor muscles in the forearm appears and lasts for some seconds. Neither fibrillation nor contraction can be induced in the upper arm or the lower limb muscles, nor in the recti abdominis.

The only somatic nervous symptoms observed in this case were fibrillary and fascicular twitching and rhythmic contractions of certain muscles. The distribution of the affected muscles was quite random, and there was no evidence of synergically associated muscles being affected. Moreover, the contractions were for the most part not spontaneous, but induced by passive manipulation of the muscles, and it seemed that as the tendency to "myoclonus" abated fibrillary and fascicular twitching took its place.

The random distribution of the clonic contractions in muscles not functionally associated, its restriction in certain circumstances to segments, strands, or even smaller muscle-fibre bundles (fascicular and fibrillary twitching), indicate very definitely that we are in all probability not dealing with a lesion of neurones of high physiological levels, but with an affection of the lower motor neurones, presumably irritative in character.

Case 6.—Delirium, Confusional State, Restlessness and Pain followed by Signs of Irritation of Lower Motor Neurones.

General symptoms.—"Influenza" followed by persistence of pains in back and limbs, fever, swelling of ankles, albuminuria and constipation.

Nervous symptoms.—(General) Headache, insomnia, restlessness, delirium and confusional state, general muscular weakness and hypotonus. (Focal) Rhythmic contractions of isolated muscles.

Gertrude B., married, aged 38. In March, 1920, patient had an illness, of which no details are available, which was diagnosed as influenza. Following this she did not recover completely, but complained of lassitude, fleeting limb and back pains, some fever at night (100°), swelling of the ankles and sleeplessness.

When seen on April 5, 1920, nothing abnormal was noted beyond œdema of the ankles and marked albuminuria.

On April 10, 1920, she became "light-headed," and complained of severe pain in the right arm, epigastrium and back.

On April 12, 1920, an erythematous rash appeared on abdomen, back and arms, with forcible twitching of muscles of neck, upper limbs and shoulders. This was accompanied by severe pain in the affected muscles. The temperature was 100° F.

On April 14, 1920, she was less restless and excited, but was very confused as to her whereabouts and did not recognize her relatives.

On April 17, 1920, the albuminuria had disappeared, the temperature remained slightly raised. The rhythmic muscular contractions persisted and she was restless and delirious at night.

She was admitted to hospital on May 31, 1920. She was pale and generally wasted and had a harassed, anxious expression. There was no trace of lethargy. She was quite disoriented as to time and place, and identified those in charge of her with various members of her family. She was inclined to be talkative and gave extravagant accounts of her doings during the day. Her mental state was like that seen in Korsakow's psychosis. Speech was normal. Hearing and vision were normal.

The pupil and their reactions were normal, the ocular movements were normal. There was no ptosis, squint, diplopia or nystagmus. All the cranial nerves were normal,

There was general wasting, weakness and flaccidity of the muscles, but no localized paresis. All movements were unsteady, probably on account of the extreme general weakness. There was no tremor. The weakness was so marked that she was very helpless and could do nothing for herself. No sensory change could be detected. The tendon jerks were all brisk, the abdominal reflexes were present and equal on the two sides. The plantar reflexes were doubtfully extensor on both sides. There was no sphincter defect.

Examination of the abdominal wall revealed a constant rhythmic contraction of the lower segments of the recti abdominis, particularly on the right side. This caused a rhythmic downward movement of the umbilicus (downwards and to right). At times, usually while she was asleep, the left sternomastoid showed a similar clonic contraction. The rate of the beats in both instances was 48 per minute. The abdominal muscle twitching undoubtedly increased in force when the muscles were palpated, and even when the patient was being examined and was consciously under observation, but it continued during sleep. It was noteworthy that if the abdominal reflexes were rhythmically stimulated at a faster rate than that of the spontaneous twitching, the rhythmic reflex responses completely replaced the spontaneous beats for the time being; that is, both movements did not occur simultaneously, one or the other monopolized the muscle.

The cerebrospinal fluid at the time of admission was clear and free from excess of cells (average of two cells per c.mm. in several counts).

The patient remained under observation for two months. During this time her mental state gradually became normal. There was at no time either lethargy or cranial nerve palsies. Her general condition steadily improved and she regained strength and weight.

The abdominal muscular twitchings became less forceful, but persisted throughout and were still present when she left hospital at the end of July.

In this case, as in Case 5, the sole somatic nervous symptoms indicated an irritative lesion of lower motor neurones. The considerations advanced in the previous instance apply here and indicate that we are dealing with involuntary muscular contractions differing in nature and site of origin from the clonic convulsive movements seen in Case 1.

Case 7.—Signs of Irritation of Sensory and Motor Spinal Roots.

General symptoms.—None observed or recorded in history.

Nervous symptoms.—(General) None observed or recorded in history. (Focal) Intense neuralgic pain in distribution of cervical sensory roots, followed by the appearance of persistent fibrillary twitching in muscles of right shoulder girdle and arm.

Charlotte M., married, aged 36. On about June 20, 1920, having gone to bed feeling perfectly well, she awoke at 2 a.m. with intense pain "in the spine of the neck" running up to the back of the head. The muscles at the back of the neck "seemed to be all on the work" and her head was drawn backwards. So severe was the pain that she had to cry out aloud.

In the morning the pain had spread to the right shoulder and seemed settled on the right side of the neck also. It was still intense and seemed to shoot outwards from the back of the neck down on to the shoulder and the upper part of the right side of the chest. She noticed that all the muscles round the right shoulder, under the right breast and in the arm as far as the elbow "were all on the twitch." Her friends "could see them wriggling through her blouse." These twitchings did not move the limb at all, but the muscles looked "as though there were worms wriggling beneath the skin." During the day the "wriggling" spread to the muscles of the forearm, and the whole limb became weak, so that she could not lift her arm up to her mouth, and her grasp became unsteady and weak. The limb felt very heavy. This condition persisted for two days and then became very much less marked, though even yet when she uses the arm they start again round the shoulder. The right arm has remained weak, she cannot hold a pen or a needle partly on this account and partly because the hand is unsteady and shaky.

The neuralgic pains persisted unabated for two weeks. During this time she could not rest for them, she slept hardly at all, and had to walk about constantly with the right arm supported to ease the pain. For the past three weeks the pain has gradually diminished, though even yet she cannot lie on the right shoulder without bringing it and the twitchings on again.

There was no lethargy at any time. There is no history of cranial nerve palsies, nor of any constitutional disturbances, such as fever, headache, constipation, nausea or vomiting.

She states that she has always been highly strung. This has been much worse since the air raids, which have made her very nervous and easily upset. Her general health has always been good. She has had three pregnancies and three healthy children born at full term. Two died during the first year of life from "convulsions," and the third is alive and well.

On admission to hospital (July 26, 1920) she was found to be a well-nourished woman of healthy appearance. Thoracic and abdominal viscera healthy. Intelligence and emotional tone normal. Vision and hearing were normal. There was no defect of cranial nerves.

The motor system: There was weakness and extreme tenderness to pressure of the following muscles of the right shoulder girdle and arm: sternomastoid, trapezius, all the scapular muscles, pectoralis major, deltoid, the rhomboids, triceps, biceps. The forearm and small hand muscles were slightly tender. There was some tenderness of serratus magnus and distinct winging of the scapula. There was general hypotonus of the affected muscles. All movements of the limb were limited in range on account of weakness, and active movements produced a tremulousness of the hand and arm that was partly due to this weakness.

Active or even passive movements of the various segments of the limb produced at once ample fascicular and fibrillary twitching of the muscles involved, and the contractions partook of the characters of both fibrillation and clonus. For although the twitchings were regular in frequency, they involved varying proportions of a muscle in different contractions, so that contractions of small muscle bundles, of strands of muscle running the whole length of the muscle and of the whole muscle simultaneously were seen. These contractions did not cause actual movement of segments of the limb.

There was no other abnormality of the musculature elsewhere in limbs or trunk.

Over the upper arm and fore-quarter on the right side there was a qualitative alteration of cutaneous sensation. All touches were felt, and pinprick was painful, but sensations appeared diffuse and not accurately localized and differed in quality from normal areas of skin. This area could not be accurately mapped out.

The tendon jerks in both arms were present and no definite inequality between the two sides could be made out. The knee and ankle jerks were normal, the abdominal reflexes present and the plantars flexor in type.

The patient was only under observation on two occasions during the last week in July and her present condition is not known.

This case did not come under observation till convalescence was well established, but nevertheless the physical signs were in accord with the history of the early stages of the illness. They were those of an irritative lesion of cervical spinal roots, both motor and sensory. If this view of the localization and character of the lesion be correct, it confirms the hypothesis that the "myoclonic" and fibrillary twitchings recorded in the two previous cases are dependent upon irritation of the lower motor neurones. In these the absence of definite tenderness of the twitching muscles and of neuralgic root pains suggests that the lesion was in grey matter, rather than in the roots.

Sicard [20] records an exactly similar case.

III.—INVOLUNTARY MOVEMENTS AND MUSCULAR CONTRACTIONS; THE SO-CALLED "MYOCLONIC" AND "CHOREIFORM" MANIFESTATIONS OF LETHARGIC ENCEPHALITIS CONSIDERED AS IRRITATIVE PHENOMENA. THE CONTRAST BETWEEN THE PARALYTIC AND THE EXCITING ACTION OF THE VIRUS.

The general impression derived from the study of this small series of cases accords with that obtained from a review of the literature of lethargic encephalitis: namely, that the constitution of the symptom-complex in any given case appears to be largely fortuitous and to follow no laws that we can yet determine. Any and every part of the nervous system may be involved and there is no combination of symptoms that we may not encounter. In consequence, many cases cannot be placed in any type unless types are to be multiplied until they cease to have either value or significance. It will be suggested later, however, that certain general principles do govern the clinical manifestations of this disease.

It is not now proposed to dwell in detail upon each of the many points for discussion arising out of these cases, and the particular aspect of the disease to which some consideration will be devoted in this paper is that introduced by the appearance of the motor symptoms mentioned in the heading to this chapter.

However, before any attempt is made to draw conclusions as to their origin and nature in the particular cases recorded here, it may be of interest to turn to the numerous published descriptions of similar cases for any useful indications they may afford in this respect. The result is somewhat disappointing, for although many comprehensive accounts are to be found, particularly in the French literature, the clinical details given are so frequently coloured by the effort to identify widely differing muscular manifestations with the "secousses myocloniques" of Sicard and Kudelski [17, 18] that their probable nature can often only be surmised.

A case reported by Roger and Aymés (16) will help to illustrate this. It is described as a "syndrôme hémimyoclonique alterne séquelle d'encéphalite épidémique."

A young adult of 22 had an acute illness with convulsions followed by the development of a right-sided hemiparesis (face, arm, leg), with inco-ordination of the right upper limb, clonic jerkings of the right arm and slightly of the right leg. The right tendon jerks were increased and the right foot showed an "intermittent" extensor response. There is said to have been spasm of the left side of the face.

It is highly probable that we are dealing here with an encephalitic lesion of the left cerebral motor cortex with Jacksonian attacks in the right limbs. Again, we find Souques [21] expressing the view that "entre l'encéphalite myoclonique et l'encéphalite choréique, il n'y a, en effet, que des différences de mesure et de rythme dans les mouvements involontaires." Achard [1] has expressed a similar view. While, finally, Comby [8] describes as myoclonic encephalitis an illness with onset of fever, malaise, torpor followed by a phase of "myoclonus" and restlessness. The involuntary movements are described as being incessant, irregular and disorderly. Here we find the convulsive movements of what are almost certainly a Jacksonian fit described as "myoclonus," the view expressed that there is no real distinction between myoclonic contractions and choreiform movements, and in Comby's paper we see the latter described under the same name as the former.

From all this it is clear that the terms "myoclonus" and "myoclonic" in connection with this disease, though precise enough in Sicard's hands, have in those of others ceased to have a specific meaning and frequently serve only to obscure the characters of the involuntary contractions and movements described in clinical reports of this disease.

In these circumstances it may be of value to consider the original meaning of the term myoclonus. It was first employed by Friedreich in 1881 to describe certain features seen in the disease he named paramyoclonus multiplex. "This affection," states Oppenheim, "is characterized by clonic contractions affecting mainly the muscles of the extremities and trunk, and rarely if ever those of the face. The contractions are short and lightning-like; they involve a small number of muscles which do not have a synergic action. The effect of the contractions in moving the limb is therefore slight or entirely absent. The contractions involve the muscles of the two sides of the body almost equally; they may be symmetrical, but not synchronous or rhythmical. The various twitchings, of which there may be sixty to one hundred a minute, are separated by intervals of varying duration. They affect a single muscle, which cannot be voluntarily contracted by itself, e.g., supinator longus. They may even be limited to parts of a muscle . . . active movements, which as a rule are unaffected, have a tranquillizing, soothing effect upon the spasm. Emotion has the opposite effect. The twitching diminishes when the attention is distracted, and disappears entirely during sleep." It may be added in conclusion that no discoverable lesion of the nervous system underlies the phenomena thus named and described.

In his first paper on the subject [17], Sicard describes the contractions in his myoclonic encephalitis as "secousses musculaires, brèves, rapides, explosives, à type rythme électrique, qui siègent sur la musculature des membres, de la face et du diaphragme, tantôt myocloniques, frappant un muscle ou un groupe de muscles, et ne s'accompagnant pas de contractions fibrillaires. Les algies disséminées persistent."

A comparison of Oppenheim's account with that of Sicard reveals the fact that the muscular contractions in the two cases bear but a relative resemblance, show certain distinct points of difference, and as far as our present knowledge goes appear to differ completely in cause.

Even, therefore, if we allow its aptness, the term myoclonus has but a limited sphere of usefulness in the study of lethargic encephalitis, and unless it is employed in Sicard's sense alone, and as a purely descriptive term without pathological basis, it had almost better be abandoned.

Nevertheless, from the descriptions given by those observers who use the term precisely we may obtain some useful information as to the probable site of the origin of these clonic contractions. Perhaps the most direct indication is to be found in two cases reported by

Sicard [17, 19], in which clonic contractions in the upper limbs gave place after several days' persistence to a paresis of peripheral type, namely, extensor weakness with wrist-drop. In one of these it is recorded that supinator longus was spared. While Sicard did not observe fibrillation associated with these contractions, such a combination has been recorded by several observers (Carnot et Gardin [6]). Again, we may note the random distribution of the clonic contractions, their frequent association with neuralgic pains of peripheral and spinal root distribution (often coinciding with that of the muscular contractions in topography). All grades of rhythmic muscular excitation have been recorded; fibrillary twitching, fascicular twitching (paramyoclonus) and contractions of whole muscles and muscle groups. These features all point to the lower motor neurone as the seat of an irritative or exciting lesion. According to Netter, "myoclonic encephalitis" marks the appearance of irritative symptoms in a disease where previously paralytic symptoms have predominated. However, neurologists have become cautious of explaining involuntary movements, and in some instances even pains, by postulating irritative lesions. Indeed, we know that the pain of the thalamic syndrome and the involuntary movements of tremor and athetosis are probably not so caused, but depend upon the release of lower centres from control. They are a reaction, an abnormal over-activity of intact portions of the nervous system when higher controlling mechanisms are out of action from destruction or otherwise. In the present instance it is extremely unlikely that we are dealing with any such release mechanism, and for the reasons given above it seems more probable that in the case of these clonic fibrillary, fascicular and muscular contractions we see the expression of an irritation or excitation of the lower motor neurone. The occurrence of Jacksonian fits indicates that the virus of lethargic encephalitis can and does act in this way upon nerve cells.

In considering the seven cases recorded in this paper, it is not proposed to discuss all the motor symptoms described, such as the Jacksonian fits of Case 1, the tremor and rigidity of Case 2, or the cerebellar symptoms of Case 4. These are in no way peculiar to lethargic encephalitis, nor does their occurrence in this disease throw any fresh light on their nature or origin. It may be of interest, however, to discuss briefly the choreiform movements of Case 3 and the fibrillary and muscular twitchings seen in Cases 5, 6, and 7.

In the preceding paragraphs and in the commentaries on the three last named cases reasons have been given for regarding the so-called myoclonic contractions and the fibrillary and fascicular twitchings with which they are frequently associated, as evidence of an irritative or exciting action of the virus of the disease upon the lower motor neurone; that is, upon the cells of the peripheral motor nerves. It is now further suggested that the extreme psychomotor excitement of certain cases of lethargic encephalitis is another example of this action of the virus, in this case upon the highest cerebral centres. If this view be correct we see in the psychical and in the choreiform motor manifestations, in the Jacksonian fits, and in the rhythmic contractions and fibrillary twitchings of various muscles and parts of muscles the expressions of irritation or abnormal excitation of neurones in each of Jackson's three physiological levels of the nervous system.

There are many reasons for regarding the choreiform agitation (the "folie musculaire" or the "syndrome choréique à grand fracas" in the expressive language of the French observers) of such a case as the third of the present series as being irritative rather than dependent upon a release mechanism. Its constant association with great psychical excitement, its frequent association with the clonic muscular and fibrillary contractions already discussed (Labbé et Hutinel [13], Sicard [20], Harvier et Levaditi [11], Ardin-Delteil et Raynaud [2], Kahn [12], Dupouy [9], and Bourges et Marcandier [4]), and its rapidly fatal termination in most instances in exhaustion, coma and death all point to its being of this nature. While the disorderly, varying and complex character of the movements not only indicates that they arise in centres of high physiological level, but also marks them off clearly from the more orderly involuntary movements of the tremor and athetosis varieties, which are accepted as depending upon removal of control over subordinate motor mechanisms.

Finally, if we can accept this view of the irritative origin of these motor phenomena another remarkable point claims attention. It is that the virus of lethargic encephalitis in its paralytic action on the nervous system has always shown a characteristic and marked selective affinity for certain groups of motor neurones in the basal ganglia and brain-stem, producing the familiar basal ganglia and midbrain types of the disease with their associated lethargy. It does not cause widespread random paralyses, such as we meet in acute poliomyelitis. In its exciting or irritating effect, on the other hand, this selective affinity is totally absent, and it seems that neurones of every level physiologically considered, or, speaking anatomically, every part of the nervous system from cerebral hemispheres to spinal roots, are indifferently attacked.

Thus the polymorphic clinical characters of lethargic encephalitis are in large measure due to this second action of the virus, and we have in effect irritative and paralytic forms or stages of the disease. Each possesses clinical features of its own. Both may be present in a given case, and they occur in numerous and widely varying combinations, or they may occur singly. However, even in purely paralytic or negative forms of the disease, the involvement of the nervous system is probably more widespread than the focal symptoms indicate. The frequent general muscular hypotonus and diminution of tendon jerks of the lethargic paralytic cases would suggest this view.

Nevertheless, the striking contrast between these two effects of the virus on the nervous system raises a question which it must remain for pathological investigation to answer; namely, whether we may not be dealing with a complex and varying virus containing more than a single active component.

IV.—SUMMARY AND CONCLUSIONS.

The protean manifestations and the remarkable clinical diversity of lethargic encephalitis are discussed, and it is suggested that the numerous schemes of classification and description adopted, though inevitable in the early and growing stages of knowledge of so polymorphic a disease, are in fact tending to confuse rather than to lend precision to our conceptions of this malady, for practically every case presents, either simultaneously or at some phase of its course, the features of several clinical "types." The example of "myoclonic encephalitis" is discussed as demonstrating the misleading results of a purely symptomatic basis of classification and description.

A natural grouping of the symptoms of any toxic infective disease of the nervous system is given, and it is suggested that description, and as far as possible classification, upon an anatomical basis—that is, using the focal nervous signs for the purpose of defining types—would obviate much of the confusion at present to be found in the literature, and would at least ensure a uniform terminology in descriptions of the disease.

Acute poliomyelitis and lethargic encephalitis are contrasted in the light of the scheme referred to.

The points thus discussed in the first chapter are illustrated by a small series of personally observed cases, and an account of various positive motor symptoms is given. It seems probable that the psychomotor excitement of the "choreiform" manifestations of lethargic encephalitis, the Jacksonian fits, and the rhythmic fibrillary, fascicular and muscular contractions collectively grouped by various writers as "myoclonic" symptoms are in fact the expressions of an irritative or exciting action of the virus of the disease upon neurones of the three physiological levels (Hughlings Jackson) of the nervous system, and that such symptoms can be localized on the same principles as are employed to localize negative or paralytic symptoms. If we accept this view of the irritative origin of the motor symptoms under discussion, a striking feature emerges: namely, that where the virus produces negative or paralytic symptoms it has, ever since the first appearance of the disease, shown a definitive and characteristic selective action on the cells of the basal ganglia and on those of certain motor nerves in the brain-stem, producing the familiar basal ganglia and midbrain types of the disease with its associated lethargy. On the other hand, in its irritative or exciting effect the virus appears to act equally on any and every part of the nervous system, from cerebral hemispheres to spinal roots, hence the polymorphic character of cases showing what we may call irritative symptoms. It is such cases that present the many difficulties in any attempt to classify lethargic encephalitis into clinical types. Possibly we may be dealing with a complex virus in which more than one active component exists.

REFERENCES.

[1] ACHARD. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 712.
[2] ARDIN-DELTEIL et REYNAUD. *Ibid.*, 1920, t. xxxvi, p. 341.
[3] BATTEN, F. E. *Brain*, 1916, vol. xxxix, p. 175.
[4] BOURGES et MARCANDIER. *Bull. et Mem. Soc. med. des Hôpitaux de Paris*, 1920, t xxxvi, p. 685.
[5] BUZZARD and GREENFIELD. *Brain*, 1916, vol. xlii, p. 305.
[6] CARNOT et GARDIN. *Bull. et Mem. Soc. med. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 125.
[7] CLAUDE. *Ibid.*, 1920, t. xxxvi, p. 269.
[8] COMBY. *Ibid*,, 1920, t. xxxvi, p. 446.
[9] DUPOUY. *Ibid.*, 1920, t. xxxvi, p. 759.
[10] ELLIS, A. W. M. *Lancet*, 1920, vol. ii, p. 114.
[11] HARVIER et LEVADITI. *Bull. et Mém. Soc. méd. des Hôpitaux de Paris*, 1920, t. xxxvi, p. 310.
[12] KAHN. *Ibid* . 1920, t. xxxvi, p. 542.
[13] LABBÉ et HUTINEL. *Ibid.*, 1920, t. xxxvi, p. 194.
[14] LEREBOULLET et FOUCART. *Ibid.*, 1920, t. xxxvi, p. 391.
[15] NETTER. *Ibid.*, 1920, t. xxxvi, p. 441.
[16] ROGER et AYMÈS. *Ibid.*, 1920, t. xxxvi, p. 244.
[17] SICARD et KUDELSKI. *Ibid.*, 1920, t. xxxvi, p. 94.
[18] *Idem. Ibid.*, 1920, t. xxxvi, p. 121.
[19] *Idem. Ibid.*, 1920, t. xxxvi, p. 123.
[20] SICARD. *Ibid.*, 1920, xxxvi, p. 260.
[21] SOUQUES. *Ibid.*, 1920, t. xxxvi, p. 552.

ON DECEREBRATE RIGIDITY IN MAN AND THE OCCURRENCE OF TONIC FITS.

BY S. A. KINNIER WILSON.

FOR a number of years certain analogies between the results of experimental mesencephalic transection in apes and other mammals and occasional clinical phenomena in man, in cases where cortical function is completely in abeyance, have appeared to me worthy of consideration. Such cases, usually of an acute nature, do not occur with any frequency, and since 1905 only some half dozen of this particular character have presented themselves, but their main features exhibit similarities so definite as at once to suggest a more or less identical origin and localization of the symptoms. Briefly, the type of case herein described and analysed is one in which there is evidence of withdrawal of cortical control in the form of unconsciousness or semi-consciousness, the result commonly of cerebral hæmorrhage—submeningeal or intraventricular, or both—or of meningeal inflammation, or from the effect of certain intracranial tumours; also, for that matter, of hysteria. Coupled with this impairment of consciousness has been the appearance of tonic rigidity of trunk and limbs, minutely resembling experimental decerebrate rigidity, to which frequent exacerbations in the form of tonic or postural fits, accentuating and exaggerating the background of tonic posture, are superadded.

It will be shown, further, that in certain types of organic and functional nervous disease these postural attitudes may be adopted involuntarily with full consciousness, and that partial or fragmentary types also occur. To this last statement much importance is attached, since the hypothesis to be subsequently developed is, that the involuntary postures of a limb or segment of a limb in many forms of nervous disease, as well as the transient positions assumed in involuntary movements such as chorea or athetosis, are in reality simply parts of the complete decerebrate attitude.

The material may be divided into two sections as follows:—

I.—(1) Cases of decerebrate rigidity or attitude and tonic fits combined.

(2) Cases of decerebrate attitude without tonic fits.

(3) Cases of tonic fits without persisting decerebrate attitude.

These three groups include only those cases where the phenomena appear in association with loss of consciousness; their significance is enhanced by the light they throw on the second section of the material:—

II.—(1) Cases of decerebrate attitude in conscious life.
(2) Incomplete, limited, or fragmentary cases.
(3) Cases of decerebrate attitude in the course of conscious involuntary movement, especially chorea and athetosis.

I wish here to express my indebtedness to various colleagues who have kindly allowed me to examine or to refer to cases which have been under their care, and to acknowledge at the outset the debt this paper owes to the genius of Dr. Hughlings Jackson and of Professor Sherrington. It has proved a matter of considerable difficulty to collate from the literature analogous cases to those herein described, mainly, no doubt, because of the usually quite incidental reference to the phenomena and the variety of the headings under which such cases might be expected to be found. Those of which a *précis* is given later provide valuable confirmation of the general views expressed in this article.

To appreciate the significance of the clinico-pathological cases more readily it is desirable to cite first the results of experimentation.

The salient features of experimental decerebrate rigidity, as contrasted with the state following on transection below or in the lower half of the medulla, are given by Sherrington [15] as follows, the latter condition being described first:—

"If in a monkey or cat transection below or in the lower half of the bulb has been performed, the animal . . . . hangs from the suspension points with deeply drooped neck, deeply drooped tail, and its pendent limbs flaccid and slightly flexed . . . . On giving the hand or foot a push forward and then releasing it the limb swings back into and somewhat beyond the position of its equilibrium under gravity; and it oscillates a few times backward and forward before finally settling down to its original position.

"To this condition of flaccid paralysis supervening upon transection in the lower half of the bulb the condition ensuing on removal of the cerebral hemispheres (mesencephalic transection) offers a great contrast. In the latter case, the animal, on being suspended just in the same manner as after the former operation, hangs with its fore-limbs thrust backward, with retraction at shoulder-joint, straightened elbow, and some flexion at wrist. The hand of the monkey is turned with its palmar face somewhat inward. The hind-limbs are similarly kept straightened and thrust backward; the hip is extended, the knee very stiffly extended, and the ankle somewhat extended. The tail, in spite of its own weight . . . is kept either straight and horizontal or often stiffly curved upward. There is a little opisthotonos of the lumbo-sacral vertebral region. The head is kept lifted up against gravity, and the chin is tilted upward under the retraction and backward rotation of the skull. . . . . When the limbs or tail are pushed from the pose they have assumed, considerable resistance to the movement is felt, and, unlike the condition after bulbar section, on being released they spring back at once to their former position and remain there for a time even more stiffly than before."

Attention is specially directed to the posture of the fore-limbs in extension-pronation, and to the head-retraction and opisthotonos.

We may now pass to an examination of cases observed in man.

I.—(1) CASES OF DECEREBRATE RIGIDITY OR ATTITUDE AND TONIC FITS COMBINED.

Case 1.—W. S., aged 19, shopman, was admitted to the National Hospital on June 19, 1905, under the care of Dr. J. A. Ormerod.

For at least four weeks previous to admission he had suffered from severe headaches, mainly frontal. About 11 p.m. in the evening of June 11, 1905, he suddenly "felt his head bad" and fell in the street unconscious, remaining so for about twenty minutes; thereafter, he complained of splitting headache across the forehead. During the night he had a fit with convulsions, but details were not forthcoming; it was followed during the next twelve hours or so by about nine other fits, apparently of a general epileptiform nature. In the course of the week before admission he frequently vomited and complained of giddiness, and was twice incontinent of urine. On getting up and attempting to walk he was stated to have staggered badly.

On admission to hospital his general condition was as follows He lay on his back in bed, frequently passing his right hand over his forehead in a half-unconscious fashion. All questions were answered rationally, but usually in monosyllables, without dysarthria. He was drowsy and indifferent to his surroundings: frequently he gave long-drawn sighing expirations: frequently, also, he would snort and spit. While he often moved the right limbs about, the left were immobile; the left arm was kept adducted and flexed at the elbow, and the left leg adducted slightly at hip and flexed slightly at knee.

Well-marked double optic neuritis was present. The pupils reacted quickly to light. There was apparently some general weakness of all ocular movements, but the patient's state did not admit of satisfactory testing. In voluntary movements the right side of the face always moved before the left. The tongue was protruded in the mid-line.

In addition to the apparent paralysis of the left limbs, it was found that the left arm showed distinct rigidity and resistance to passive movements, and the left leg similarly. No sensory changes were detected. The arm jerks were equally diminished on the two sides. The abdominal reflexes were much down on the left. Knee and Achilles jerks were diminished on both sides, and a well-marked double extensor response was readily obtained, but no ankle clonus could be elicited. Occasional incontinence of urine was noted.

By lumbar puncture 20 c.c. of fluid were drawn off under considerable tension, pale, reddish-yellow in colour, quite translucent, and obviously hæmorrhagic. There was no difference between the first drop and the last.

The next day the patient was able, after much encouragement, to move the left arm a little at shoulder and elbow, and to make a feeble grasp with the left hand. On the other hand, the facial asymmetry on volitional movement was accentuated. Ocular movements were still imperfect; the sixth nerves were apparently weaker than the others, slight internal strabismus being noted with the eyes at rest. The knee-jerks were further diminished, especially the left, and the double extensor response persisted.

A diagnosis of cerebral tumour was made, probably right frontal, and, further, it was presumed that for some reason slight hæmorrhage into and from the tumour had taken place when the symptoms had become acute just prior to admission.

At this point, however, they changed, and presented the following striking clinical picture.

On the same day, June 23, 1905, at 11.10 p.m., I was called to see the patient.

He was lying on his back, with head turned slightly to the right, and was quite unconscious. The arms were extended by the sides, slightly flexed at the wrists, and notably rigid. The legs were also extended and adducted, with toes pointed down and in. Respiration was extraordinarily laboured, the rate no more than five to the minute; inspiration was short, about three seconds' duration, while expiration was long drawn out, averaging nine seconds. It was noisy at first, gradually becoming fainter and fainter, then a sudden jerky inspiration completed the cycle. The pulse was 180, full, of moderate tension, and slightly irregular, sometimes thudding for several quick beats, then intermitting. With inspiration it always beat more quickly and strong.

The pupils were regular and equal, about 4 mm., and reacted to light. The corneal reflexes were absent. When the lids were lifted the eyes were seen to be fixed and staring straight in front. Skin and mucous membranes were flushed but not cyanotic.

11.15 p.m. Respiration remained about 6; pulse had fallen to 96, and was again regular. The knee-jerks could not be elicited on either side, but a smart patellar tap produced in each case a contralateral adductor jerk. The legs remained fully extended and rigid. The recti abdominis were also rigid Patient was incontinent of urine, and a minute later had an erection.

11.20 p.m. The eyes began to turn slightly downwards, and at the same time the right pupil dilated till it was almost twice the diameter of the left; it was now immobile to a bright light, though the left contracted as before. The plantar responses, up to this point absent, were now extensor on both sides.

11.30 p.m. The eyes gradually deviated into a definite and unmistakable skew-position, the right eye being down and in, the left eye up and out. Both pupils reacted again to light, though the right remained larger than the left. The skew-deviation was maintained only for a minute and a half, both eyes then slowly moving to a more symmetrical position, while the right pupil became of the same size as the other.

11.45 p.m. About this time both arms became rigid, the left rather more than the right. Both were adducted at the shoulder and strongly extended at the elbow, with pronounced internal rotation and hyperpronation; the wrists were flexed, so that the palms of the hands looked up and out, quite turned away from the body (fig. 1). This striking position of the arms was henceforward maintained; at intervals the whole arm would stiffen still further, as it were, and the attitude become more accentuated, as if by waves of contraction passing down the musculature, but even when these waves relaxed the extensor-pronation position was not abandoned. The legs were as before, rigid, adducted, extended, feet inverted and great toes strongly dorsiflexed.

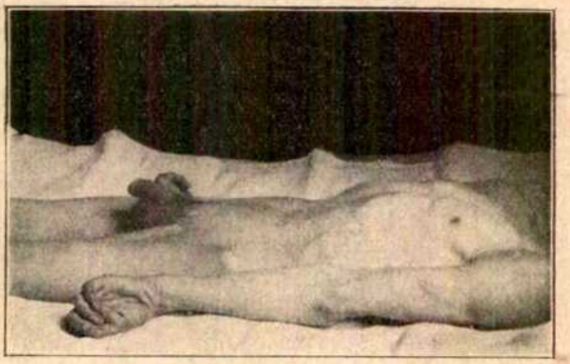


FIG. 1 (Case 1).—The typical decerebrate attitude of extension-pronation.

12.20 a.m. Another fit started. Patient suddenly became absolutely stiff in the decerebrate position, and his face reddened, while respiration became occasionally laboured, down to four per minute. It consisted of a short inspiration followed by a long-drawn-out expiration in the proportion of about 1 : 4. The pulse had become much more rapid again, 140 in rate, but it was fairly regular. There was no twitching of the limbs, but simply an intense tonic spasm accentuating the extensor attitude. The eyes were open, staring straight in front; pupils were dilated to their widest and did not react to light; corneal reflexes were absent on both sides.

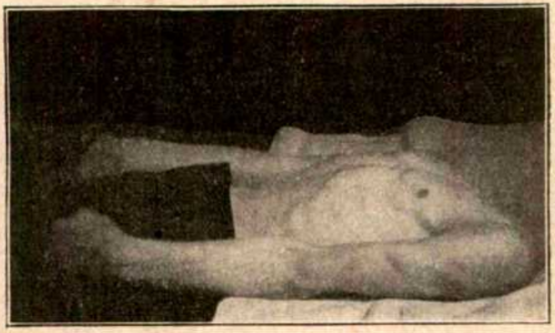


FIG. 2 (Case 1).—The typical attitude at the summit of one of the actual tonic fits. The left forearm is less pronated than the right. Intense tonic contraction of the extensor musculature, with slight wrist flexion.

[For figs. 1 and 2 I am indebted to Dr. Colin Russel, of Montreal, who was in residence with me at Queen Square at the time.]

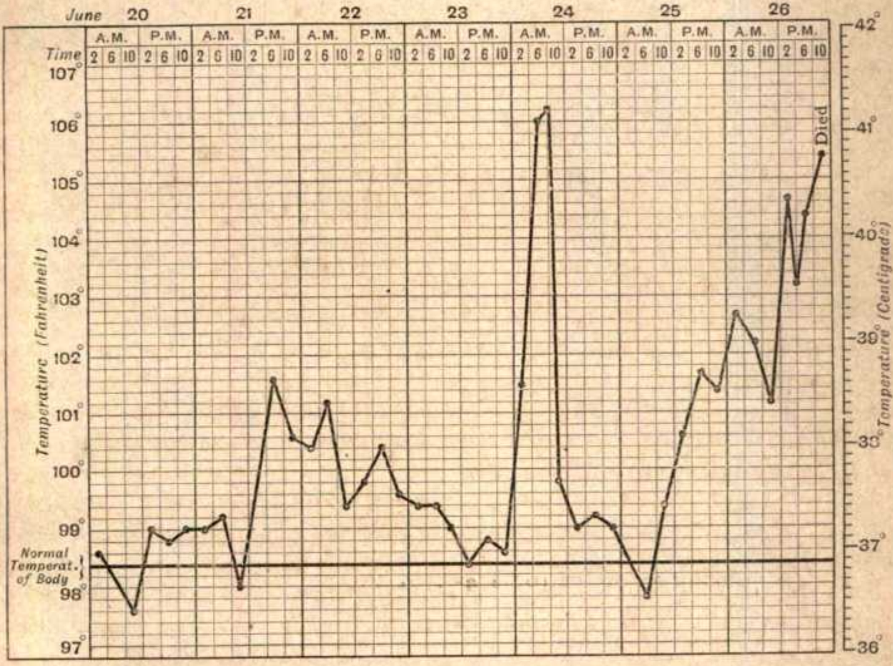


FIG. 3.

1.25 a.m. When the patient was seen again, after about an hour's interval, he was in the same extensor position, arms adducted at shoulders, extended at elbow, flexed at wrist, whole arm rotated inwards and hyperpronated; on the right the thumb was between the first and second fingers of the hand, but not on the left (fig. 2). The legs were rigidly extended, feet inverted, great toes dorsiflexed. Respiration 9, pulse 130.

For the remaining two days of the patient's life the phenomena continued at very frequent intervals without cessation. It appeared that any handling of the patient, e.g., during sponging (for with the decerebration high irregular temperature occurred, see fig. 3), precipitated the "attack," which consisted, approximately, of a period of Cheyne-Stokes' breathing for about one minute, associated with intense extensor rigidity of all limbs, inversion and hyper-pronation of arms, flexion of wrists, thumbs between first and second fingers. The stiffening of the limbs preceded the onset of the altered breathing by a second or two; the face flushed slightly; the eyes always opened widely and the pupils dilated. In the feet there was usually flexion of the small toes, while the great toes were dorsiflexed proximally and flexed distally.

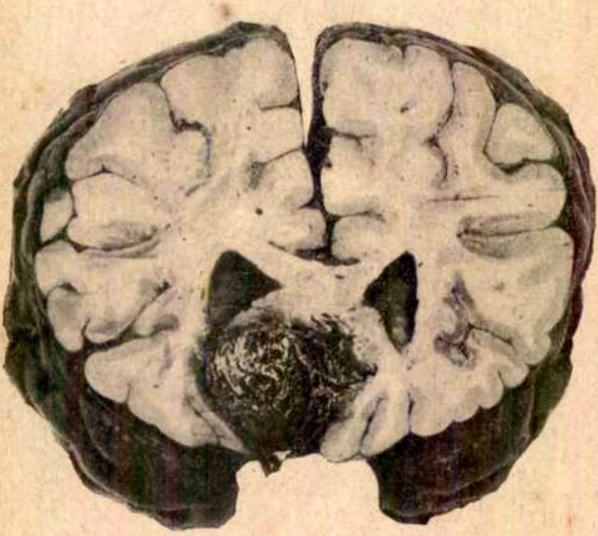


FIG. 4 (Case 1).—Position of vascular glioma (right frontal), whence hæmorrhage spread through the entire ventricular system.

Death ensued after almost exactly seventy-two hours of complete decerebrate rigidity, with many scores of tonic fits. Post-mortem, a vascular glioma was found on the under-surface of the right frontal lobe (fig. 4), from which hæmorrhage had taken place, making its way right through the ventricular system and under the basal meninges, and spreading beyond the cranial cavity to the spinal theca. The third and fourth ventricles were blocked with blood.

The phenomena exhibited by the patient during these hours resemble in minute particulars those of mesencephalic transection in experimental animals. Until the hæmorrhage and increasing pressure killed him he was little or no more than a decerebrate preparation.

Case 2.—L. C., aged 34, a soldier, was admitted to hospital at 10 p.m. on the evening of January 29, 1918, with the history of having had a "stroke" earlier in the evening.

On admission he was conscious, and was able to speak fairly clearly. The left face, arm, and leg were not moved voluntarily. Complaint was made of severe pain in the head, which he frequently rubbed with his right hand. The pupils were of medium size, equal, and reacted to light. The mouth was drawn slightly to the right. All the deep reflexes were brisk, the knee-jerks excessively so, and there was double ankle clonus, with a double extensor-plantar response.

About 1 a.m. the patient suddenly became quite unconscious, and shortly after a series of convulsive attacks commenced and continued uninterruptedly for many hours, till within a short time of the patient's death the same day, January 30, at 2.25 p.m.

These attacks were of the following nature :—

The whole of the musculature of trunk, arms and legs became rigid ; the arms and legs were extended to the fullest extent, the arms by the sides and the legs together. In addition, both arms were strongly inverted and hyperpronated, while the hands were clenched and the wrists slightly flexed. The legs were slightly inverted, the feet more notably so, with the toes pointing down and in. It was remarkable that notwithstanding the pre-existing paralysis on the left side, the musculature of both sides was equally and symmetrically involved in the tonic fits. While the musculature was in this rigid condition, fresh "waves," as it were, of tonic contraction passed through it, accentuating for the moment, if that were possible, the already existing posture.

The duration of each attack was less than half a minute. All through the night and far on into the next day these tonic fits continued, the patient never regaining consciousness. The interval between each was simply a few minutes.

At the autopsy recent fluid blood was found exuding from the pial roof of the fourth ventricle and spreading over the corpus callosum and over the whole of the basal surface of the hemispheres. On section an extensive recent hæmorrhage was seen to have ploughed up the interior of the right cerebral hemisphere and to have burst into the lateral ventricle, and so to the opposite lateral ventricle and right down the chain of ventricles to the fourth and through the foramina in its pial roof. The blood had also passed down the surface of the cord. There was obvious syphilitic disease of the cerebral vessels, and a small unruptured aneurism was found on the right middle cerebral artery. The free ruptured ends of the lenticulo-striate set of vessels on the right side lay in blood clot, but no aneurisms were found on them.

Case 3.—L. F., aged 39, married woman, was admitted to the National Hospital, under the care of Dr. James Taylor, on January 27, 1905.

In July, 1904, the patient began to suffer from severe headache and pain in the left occipito-parietal region, with giddiness.

In January, 1905, some weeks before admission, she noticed that her articulation was becoming defective, and within a few days the right arm and leg became distinctly weak, and the seat of occasional paræsthesiæ.

On her admission, moderate optic neuritis of both discs was found. There was well-marked weakness of the right side of the face, for both voluntary and involuntary movements, though less for the latter than the former. The patient lay in bed with the face turned to the left, the chin slightly up, the occiput to the right and rather down towards the right shoulder. The right arm was closely adducted to the chest, elbow at a right angle, forearm incompletely pronated, wrist extended, fingers and thumb slightly flexed. The whole limb was in a state of extreme hypertonus, and only slow and very weak voluntary movements were obtained in the muscle groups. The right leg was fully extended at hip, knee, and ankle, strongly hypertonic, and immobile.

A very brisk jaw-jerk was obtained, and though the patient did not complain in any way of the left limbs, and their volitional power seemed unimpaired, yet both abdominal reflexes were absent and the plantar response was definitely extensor on both sides.

On the fourth day after her admission a series of clinical phenomena of an unusual nature commenced and continued till her death five days later.

It was observed on the forenoon of January 31 that the patient was becoming drowsy and at one o'clock the left arm and leg suddenly passed into a rigid tonic spasm and relaxed again after five minutes. A similar tonic spasm of the left side occurred at two o'clock, of about the same duration. At six o'clock she was absolutely unconscious, the pupils widely dilated and immobile to bright light, respirations so shallow and noiseless as to be almost imperceptible, rate 16; pulse only 42, and slightly irregular. The patient lay immobile on her back, with limbs fully extended, and unexpectedly limp, only the slightest resistance to passive movement being noted in the joints. Within a brief period, however, respiration suddenly became noisy, irregular, and cogwheel or sobbing in character, and at the same time the extended limbs passed into a state of extreme rigidity in the decerebrate attitude; the upper extremities were adducted and internally rotated, the forearms strongly hyper-pronated, so that the backs of the hands faced each other; trunk and neck were extremely rigid, and the latter slightly retracted; the legs were similarly in full extension, heels drawn up, toes pointing down and feet inverted. From time to time during the next few hours the rigidity waxed and waned, within moderate limits, but by 1.30 a.m. it ceased and the patient's limbs became once more relaxed, though the attitude of extension and pronation was unaltered.

During the next day the same alternation of tonic fits of all the muscles of the body with relaxation of tone occurred at intervals. That morning, when the mouth was widely opened with a gag, it was seen that the uvula and soft palate were the seat of a rhythmical contraction and relaxation at the rate of about 120 a minute, entirely symmetrical on the two sides, there being no deviation of the raphe. This so-called "palatal nystagmus" continued without break or intermission for hours, and was re-observed at intervals all day. It is worthy of special attention.

On February 4 the limbs were flaccid and the patient apparently lifeless, respiration being imperceptible. Yet the extensor attitude never changed.

Death ensued the same day. At the post-mortem examination a tumour of the mesencephalon was discovered, invading the upper part of the pons. It proved to be a glioma.

Case 4.—M. B., a little girl, aged 6 years, was admitted to the National Hospital under my care on March 13, 1920.[1]

The history as given by the child's mother was that some weeks previously the patient had caught a cold, become rather deaf, and developed earache, which was followed a week later by headache, vomiting, and a rise of temperature. After three days of high fever her condition rather improved, but the headache and the vomiting persisted; actual otorrhœa never occurred. During the week previous to admission the symptoms became aggravated, and mild delirium set in, with restlessness and talkativeness. A squint also developed. The child was practically unconscious on admission to the hospital.

Neurological examination revealed hyperæmia and some blurring of the discs, and bulging of the tympanum of the right ear. Almost complete right and incomplete left ophthalmoplegia, interna and externa, with weakness of the left side of the face, was noted.

The child lay in a semi-conscious state with eyes half closed, mouth open, and head turned to the left. The left arm was flexed acutely at the elbow, adducted at the shoulder, and extended at the wrist, the fingers being flexed; the right arm lay in an easy extended position; the legs were fully extended and adducted, and the toes pointed downwards (fig. 5). From time to time the child moved her limbs in a vague and restless way, moaning the while, and occasionally opening her eyes.

At intervals during the twenty-four hours of her life which remained she exhibited tonic fits in a highly characteristic form. These were most commonly bilateral, but occasionally more severe on the left side than on the right.

A brief period of excitement and moaning ushered in the attacks, which consisted of sudden powerful opisthotonos and head retraction, the neck straightening and the occiput nearly touching the shoulders. The left arm relinquished its flexed attitude and became strongly extended, the forearm over-pronated, the wrist extended to a complete right angle, the first and second fingers extended and the third and fourth flexed, while the thumb was flexed

---

[1] For the notes of this case I am indebted to my house physician, Dr. M. A. Blandy.

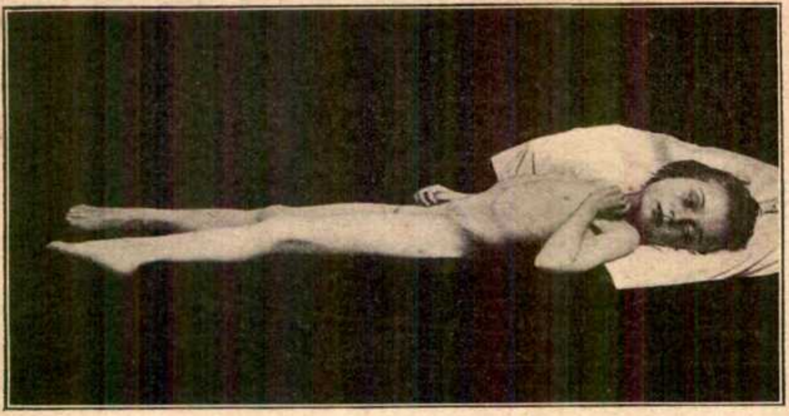


FIG. 5 (Case 4).—Incomplete decerebrate attitude in the intervals between the tonic fits. On the occurrence of the latter the left arm passed to full extension-pronation while the head straightened and was mesially retracted.

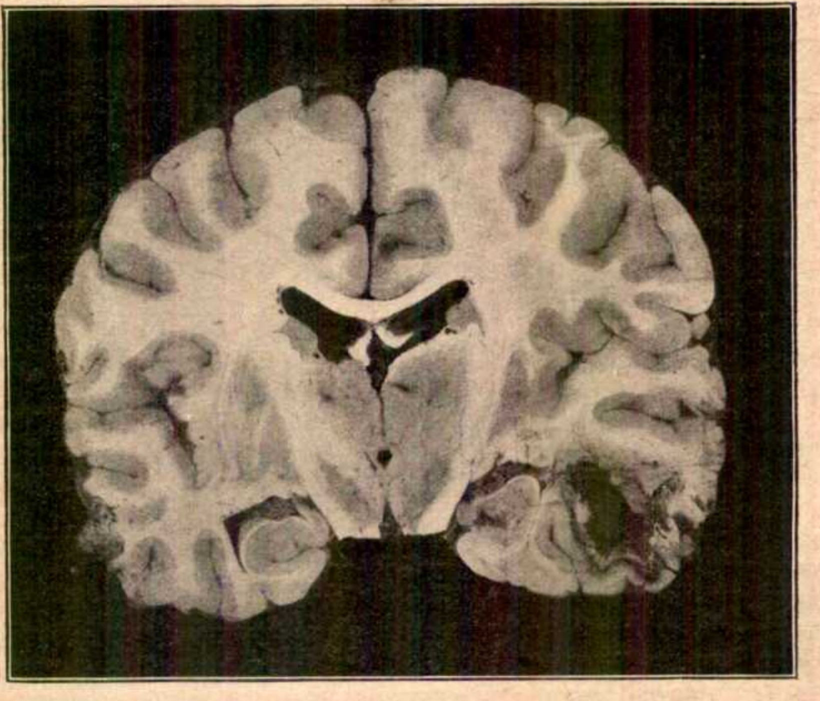


FIG. 6 (Case 4).—Shows position of temporal abscess from which the ventricular system became infected.

into the palm. The legs were in fullest extension with heels up and toes down, and the right arm was as the left, except that it was rather less inverted and the hand was in the form of a fist. Trunk and limbs alike were absolutely rigid. During the attacks, which lasted about ten to fifteen seconds, respiration quickened, and was accompanied by slight expiratory groaning. No clonic movements occurred at any time in the course of the attack, cessation of which was followed by relaxation of the hypertonicity and resumption of the original attitude described above.

Examination of the reflexes showed absence of the abdominals, activity of all the deep reflexes, bilateral extensor plantars, and a crossed extensor from the right to the left.

Lumbar puncture revealed a somewhat turbulent cerebrospinal fluid, in which organisms of the streptopneumococcus class were found. Death ensued within thirty-six hours of the patient's admission.

At the post-mortem examination there was general meningitis over the cortex and more marked along the base. The right middle ear was full of pus. Section revealed an abscess in the right temporosphenoidal lobe, whence, apparently, the ventricular system had become infected (fig. 6). There was but slight internal hydrocephalus.

Case 5.—M. C., aged 2¾ years, was admitted to the National Hospital under Dr. Ormerod on February 28, 1906.

Three weeks before admission the little girl had had a generalized epileptic attack, followed by another an hour or so later, Since, she had been apathetic yet irritable, occasionally giving vent to a cry. She had also become inattentive to her excretory functions.

On admission, the patient was drowsy but irritable, lying more or less constantly on her side with complete flexion of all her limbs, and with her back bent, so that she was literally "huddled up" in bed. The limbs could be passively extended at all joints, but the procedure brought out a certain amount of resistance in the flexors from hypertonus. The knee-jerks were active and the plantars flexor.

March 3, 1905: Patient comatose and roused with great difficulty. Occasional short spasmodic flexion movements of all limbs. Temperature 100·8° F.

10.30 p.m.: The arms remained flexed at all joints, but the legs had become fully extended at hip, knee, and ankle. Abdominal reflexes absent, knee-jerks active, double extensor response.

March 4, 1906: The extensor position of the legs was not maintained; they were sometimes flexed, sometimes extended.

March 6, 1906: The legs were now extended again, and so were the arms. The patient lay unconscious, with the arms extended by the sides and the forearms notably hyperpronated. The hands were clenched. The legs were also fully extended, with feet inverted and toes pointing down and in.

At frequent intervals tonic spasms passed down the musculature, reinforcing and accentuating the persisting extensor attitude of arms and legs. It was noteworthy that in these brief attacks the pronation of the forearms was always markedly increased. The trunk was extended, but not to an opisthotonic degree, nor was there more than slight retraction of the head.

This extensor rigidity, with occasional increase from transient extensor and pronation spasms, was maintained unchanged for the next few days, in fact up to the patient's death on March 11, 1906 (fig. 7).

Post-mortem: Typical tuberculous meningitis of the base, with moderate internal hydrocephalus.

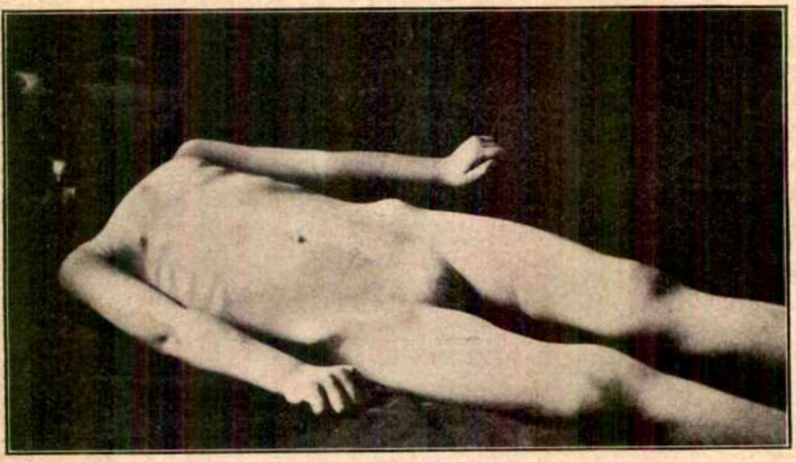


FIG. 7 (Case 5).—Decerebrate attitude maintained for several days *sub finem*.

A consideration of these five cases will suffice to illustrate the cardinal features of decerebrate rigidity in man, in its complete form. In each the decerebrate posture is reproduced with the exactness of a physiological experiment, and once it is appreciated it can always be recognized for what it is. The details of the attitude in man being on a larger scale, can be studied with greater ease than in mammals. One of its most significant components is the extension-pronation of the arms and the flexion of the wrists. We shall see later how it is just these components which are most likely to make their appearance in incomplete cases. The reader will also note that in all five examples the onset of tonic fits merely accentuated the already existing posture, showing that tonic fits are in reality attacks of decerebration, if the expression be allowed. The absence of any clonic element is of much significance. It may be taken to indicate that tonic and clonic movements have different origins and are the expression of the activity of different motor mechanisms; one is characterized by phasic and the other by static activity.

From the pathological viewpoint it will already be apparent that in each instance the morbid process was so situated as to dissociate the cortex functionally from lower centres, which exhibit an activity of their own until such time as they themselves are overwhelmed.

It is particularly noteworthy that in many of the cases striking respiratory disorder ushered in the tonic attacks or persisted with the maintenance of the decerebrate posture.

(2) Cases of Decerebrate Attitude without Tonic Fits.

Case 6.—I owe this case to the kindness of my colleague at King's College Hospital, Dr. G. F. Still.

D. S., aged 15 months, was admitted to hospital, under the care of Dr. Still on February 28, 1920, with a history of "being ill" for six weeks. The chief clinical symptom was persistent vomiting after every meal for the previous three weeks. It was noted by the mother on the day of admission that the child's eyes seemed "fixed" and that its limbs were sometimes trembling and sometimes stiff. No fit had been observed, but for a day or more the infant was thought not to have recognized its mother.

On admission the optic discs were normal, the pupils reacted rather sluggishly to light, the fontanelle was flat. The child lay in an apparently unconscious condition with arms and legs extended and rigid, but without head-retraction. Kernig's sign was not obtained. From this date to the child's death, six days later, the attitude of decerebrate rigidity was maintained and indeed intensified. Thus the arms were fully extended, adducted, strongly hyperpronated; the backs of the hands faced each other, and at the same time the wrist and finger flexors were conspicuously contracted; the neck was stiff from muscular rigidity but was not retracted in any degree; the lower extremities were in full extension and adduction, with heels drawn up, toes pointing down, and feet slightly inverted (fig. 8). The abdominal reflexes could not be elicited, the knee-jerks were active and the plantars extensor. With a pulse of 150 was coupled a slow, sighing respiration. The child died quietly on March 9, after unconsciousness and decerebrate rigidity of some nine days duration.

Permission for a post-mortem examination was refused.

(3) Cases of Tonic Fits without Persisting Decerebrate Rigidity.

Less infrequent, possibly, than cases belonging to either of the two previous categories are those in which the occurrence of tonic fits is not associated with any maintenance of a decerebrate-rigidity attitude apart from what obtains during the fit itself. "Cerebellar fits," so-called—not the best name to give them—have long been known, and since the days of Hughlings Jackson little has been added to his descriptions and interpretations. It is, however, desirable, not merely to bring the phenomena of tonic fits into line with what experimental physiology has taught us of decerebrate rigidity, but also to show that *it is possible to resolve these complete seizures into component elements, that partial, incomplete, even segmental tonic attacks may be met with, and that some at least of the more or less fixed attitudes of head and neck, trunk, and of one or other limb or part of a limb, encountered in certain basal, subtentorial and other lesions, are in reality functional fragments, so to say,*

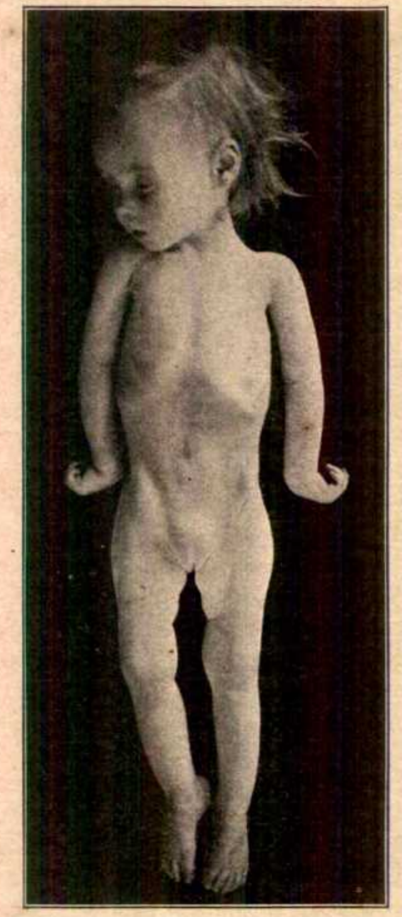


FIG. 8 (Case 6).—Striking decerebrate posture of limbs and trunk, but with no head retraction. Photo taken from above, with child lying on back.

of a non-cortical motor mechanism which finds its full expression in decerebrate rigidity and in complete tonic fits.

For the moment it is immaterial to our purpose whether these "tetanus-like seizures," as Jackson called them, are of organic or hysterical origin; a number of instances of each have come under observation.

Much the most striking of all is the following case, difficult though it be of exact diagnosis.

Case 7.—V. S., aged 18, was admitted to the National Hospital under the care of the late Dr. C. E. Beevor, on April 10, 1906.

Some months previously he had fallen down a flight of stairs on to his head, and though momentarily stunned and thereafter dazed for some hours he had not, apparently, really lost consciousness. Two weeks later he was thrown down some stairs, apparently in a "rag," struck his head again, and lost consciousness, but it is not known for how long. Thereafter he complained of pains in the head, shakiness, and drowsiness. Two months later he had an attack of influenza, and two days thereafter his first fit. These were of a peculiar character, described in detail below, and recurred on an average three or four times a week. They presented certain diagnostic difficulties, it is true, and opinion was divided as to their organic or hysterical nature, but it is important to note that the patient had had the attacks when in company and when alone in bed, indifferently, and that during his stay in hospital the efforts made to inhibit or cut short the attacks by the usual methods adopted in cases of hysteria were on each occasion a conspicuous failure.

On objective examination no signs of any organic disease of the central nervous system were found. The optic discs were clear and the reflexes in all respects normal.

The following is an account of a series of fits observed by myself.

When I reached the patient he was already lying in the semi-conscious condition succeeding a fit. He was breathing heavily, foaming slightly at the mouth, and was on his back. A moment later, he passed into a fit.

His arms went out in front of him, extended and hyperpronated, with hands clenched and wrists flexed. The eyes rolled up and the eyelids closed. The face appeared drawn, as if in a tonic spasm, while the arms and to a less extent the legs were absolutely stiff. Then the arms went out from the sides, and he began to rotate from left to right, so that he turned right round and lay on his face. This turning movement was slow and deliberate, and it was accompanied by slow stertorous respiration. At the same time as he turned his body became slightly curved anteroposteriorly, with the concavity forwards. He did not turn any further, but lay thus on his face. There was no clonic movement of any sort. Then in a few seconds came a relaxation, whereby the tonic spasm died down, and the limbs became completely atonic, flabby, so that they could be shaken about like flails. His respiration became more easy, and his colour less congested.

Nurse and I turned him round on to his back again.

A moment later, he passed into a second fit.

His corneal reflexes, tested at the commencement of this, were absent. The eyes deviated strongly upward, and at the same time the left arm went out into a tonic spasm, being away from his side and hyperpronated. At the same moment as his arm went out, he arched his body backwards, so that the occiput was down on the nape of the neck, and the muscles of the back in a powerful tonic contraction. The right arm was by the side, and it then contracted strongly in extension and internal rotation. With this, he again began to rotate, the left arm leading the way, and the trunk coming round after it from left to right, until he had come round on to his back again, nurse helping him, otherwise he would have been off the bed.

The same sequelæ, relaxation of the contractions and complete atonia, were observed. In the interim, the corneal reflexes returned, but he made no response to stimuli.

He then passed into a third fit.

This began by his rising off the bed completely, owing to strong contraction of the recti abdominis, &c., and in this anteroposterior plane he curved right forwards. Then his arms went away from the sides, the left before the right, both being powerfully rotated internally, and the hands clenched. The fists were strongly flexed on the forearms. The eyes were closed and the face drawn and quivering slightly. The under lip was curved, the corners of the mouth being depressed.

The position just mentioned was maintained for a moment, then he began to rotate slowly to the right, and at the same time to arch back into an opisthotonic position. A moment later, therefore, he was on his left side, his arms as before, his back most powerfully contracted, the head right back, the face looking upwards. Round he came still further, till he was on his face, and as he was breathing heavily we turned him on to his back again. There was no clonic movement whatever. I tried his jerks rapidly, and found they were all increased, the abdominals were diminished, especially on the left side, and there was a double flexor response. There was no incontinence and no biting of the tongue.

He sank a few seconds into the same atonic condition, with increased jerks, and lay motionless, salivating slightly.

Almost as soon as he had come round on his back the muscles relaxed and the limbs again became peculiarly atonic. He breathed deeply and his colour improved. There was no extensor response, but both abdominal reflexes were diminished.

A minute or two later, he put out both arms by his sides, separating his fingers, and then opened his eyes widely. His respiration quickened and he smacked his lips a little.

In this remarkable series of absolutely pure tonic fits, whatever their pathogenic basis, it is plain that the postures adopted were for the major part precisely those of decerebration, especially in regard to the extensor rigidity with pronation of forearms and flexion of wrists, and the head retraction and opisthotonos. The changing nature of the movements, notably the occasional trunk flexion and the rotation, indicates that in persisting decerebrate rigidity the posture may be in part compounded of mutually antagonistic couples, which may be dissociated in the course of an actual tonic fit, and in part the result of one of these being for some reason overborne by the other. Reference is made to this consideration in a subsequent section. Where there is no persisting *structural* cortico-mesencephalic separation there is less likely to be fixity of posture.

Of several other cases of tonic fits associated with unmistakable organic disease the following may be selected as sufficiently illustrative.

Case 8.—The patient was a woman of 28 with a comparatively short history of headache, giddiness, vomiting, and optic neuritis. These general symptoms of intracranial tumour were not, however, accompanied by any definite localizing sign, although the persistent pain over the left side of the head warranted a left occipito-parietal decompression. During the five days between the operation and the patient's death she had several fits of a strictly tonic nature.

She suddenly became unconscious while the head wound was being dressed, both legs going out into strong extensor tonic spasm, the knees adducted and the feet powerfully plantar-flexed and inverted: the arms were similarly extended and adducted, with forearms hyperpronated on one occasion, but on another definitely rotated outwards, i.e., in a position of supination; the fingers were strongly adducted and extended, while the thumbs were flexed at the metacarpo-phalangeal joints and adducted across the palms. The head was markedly retracted and there was a distinct degree of opisthotonos of the spinal column. During the attacks, which lasted not more than 30 seconds, respiration always became very irregular. Immediately after the cessation of the tonic stage the limbs relaxed, seemed empty of tone, so to say, and could be moved about like flails.

At the post-mortem examination a glioma, of the size of a walnut, was found invading the cortex and white matter of the middle lobe of the cerebellum towards its left side, and of part of the left lateral lobe. From the operation wound and subcortical exploration then made blood had oozed into the left lateral ventricle and passed downwards.

It is obvious that the presence of the tumour alone will not explain the generalized bilateral tonic fits, and I attach more pathogenic importance to the results of the operative interference and the intraventricular hæmorrhage.

The occurrence of tonic fits as a hysterical manifestation is a neurological platitude, but their real significance for the purposes of this paper consists less in their psychogenic origin than in the morphological aspect they may assume. If we imagine the most superficial degree of hysterical dissociation to involve the transcortical neuronic level the movements released will be purposeful and complex, resembling normal motor activity except for full conscious participation therein; if, similarly, we assume functional disintegration to descend to lower neuronic levels we should expect to observe phenomena morphologically analogous to those of decerebrate rigidity, which is, as a fact, precisely the case. In this way the actual movements of certain moderately severe hysterical seizures receive a physiological explanation and illustrate the disorders of function which we have associated with organic decerebrate human preparations.

It seems unnecessary to cite any case in support of this contention, so commonly are the opisthotonic and extension-pronation postures found in hysteria. The reader is referred to one of Richer's drawings,[1] from which it will readily be seen that the attitude of the *arc de cercle* is strictly comparable to that following mesencephalic transection.

Scattered through the literature are a fair number of cases of what should be called "decerebrate rigidity in man," some of which have been observed with sufficient minuteness to be of documentary value. Of older cases, that recorded by Bastian [1], under the caption: "A Case of Apoplexy in a Boy, aged 15; Intraventricular Hæmorrhage, Convulsions, and Death in Four Hours," may be mentioned, and there are, of course, the original cases of Hughlings Jackson [11], some of which were republished in *Brain* (1906), and several to which he himself referred (Dreschfeld, Drummond, Bacon). One of the cerebellar cases of Stewart and Holmes [16] (No. 9 in their paper) is of particular value in this connection, and the recent article by Douglas Firth [6] may also be referred to. A good example of tonic fits in association with a large cyst of the cerebellum was reported by Eskridge [5]. Of particular interest are two papers by Bruce, one entitled: "A Case of Intracranial Aneurism in a Young Subject" [3], and the other, "Aneurism of the Anterior Cerebral Artery with unusual Prolongation of Life after Rupture; Autopsy" [4], and a third, published by Dr. Violet Turner [19], under the heading, "A Case of Prolonged Hyperpyrexia in a Child with a Mid-brain Tumour," of which three cases a précis follows.

---

[1] Paul Richer: "Etudes cliniques sur l'hystéro-épilepsie." Paris, 1881. Plate III, fig 2.

(1) *Bruce* (1904).

The patient was a miner, aged 27, whose chief complaint was of absolutely unbearable headache, but apart from slight congestion of the optic discs no objective neurological sign was discovered on minute examination. Ten days later he developed paresis of the right third and sixth cranial nerves. After temporary improvement, a fortnight from his admission the symptoms suddenly became acute again, and at 8.20 one evening he had a general epileptic fit, from which he never recovered consciousness. A second convulsion occurred at 9.15, and a third at 9.50. During the night numerous attacks of spasm occurred. For some hours these occurred about every five minutes, each period of spasm lasting about forty or fifty seconds. The spasm began with a gurgling sound in the throat; the breathing became accelerated (to 52 per minute) and noisy; and the pulse to 120 or 130. *The limbs became rigid: the arms were extended in front of the body and rotated so that the hands were placed back to back. The fingers were closed.*

The intercostal muscles and the diaphragm acted violently. In the intervals between the spasms the breathing was comparatively quiet and tranquil, and the limbs were placid. The pupils varied in size and reacted sluggishly to light. The patient died at 7 a.m.

At the post-mortem examination the base of the brain was covered by extensive diffuse subarachnoid hæmorrhage, spreading from the frontal region, optic chiasma, anterior surface of the pons and medulla, to the cerebellum. On opening the brain there was found a considerable quantity of recent coagulum in both lateral ventricles, but more abundant in the left. On separating the frontal lobes there was found, arising from the anterior communicating artery, a spherical aneurism about the size of a small hazel nut. It had burst on the left side and there was considerable softening and infiltration of the tissues of the left frontal lobe. The blood had passed both upwards into the ventricular system and into the subarachnoid space, where it had become widely diffused.

(2) *Bruce* (1908).

The patient was a smith, aged 33, who was admitted to hospital in a state of unconsciousness, which was said to have lasted about two hours.

On admission, he lay with arms and legs extended; the eyelids were half closed, the pupils contracted, circular, equal in size, and reacted to light only to a slight degree, if at all. Breathing was irregular both in rhythm and in depth of inspiration. The arms lay along the sides of the trunk, but *there were frequent spasms in which they were forcibly extended at the elbow and drawn to the side of the chest, or even in front of it. The forearms were very forcibly pronated, so that the backs of the hands were directed towards each other. The wrists were flexed, and the fingers were fully flexed, causing the hand to be tightly clenched, the thumbs being strongly adducted against the index fingers. During these tonic spasms the lower extremities were tonically extended, and* *there was extension at the ankle joints and inversion of the feet.* The spasms lasted a few seconds, and were usually accompanied by a loud expiratory effort and puffing out of the cheeks, but the facial muscles were otherwise not involved. The head tended to rotate to the left side, and the chin was approximated to the left shoulder.

There was ankle clonus on both sides, but both plantar reflexes were flexor in type.

During the day the spasms continued. By 3.30 *the respirations were more definitely of a Cheyne-Stokes character, and at the height of the cycle the arms and legs became rigid and the back tended to be arched.* Lumbar puncture was performed, and 25 c.c. of a brownish red fluid were drawn off under high pressure. This fluid was obviously composed of blood and cerebrospinal fluid mixed. By 8 p.m. the spasms had ceased.

The patient lingered in a comatose state for eleven more days, when he died.

At the post-mortem examination a small aneurism, the size of a pea, was discovered on the left anterior cerebral artery. It had given way, and from it the blood had spread directly into the subarachnoid space, passing along in all directions, filling up the sulci and the subarachnoid cisterns, to some extent "seeking an entrance" into the fourth ventricle from the foramen of Majendie, and also passing along the spinal cord under the arachnoid. The blood had also ploughed right through the corpus callosum and had entered the lateral ventricles and had coagulated there.

(3) *Turner* (1916).

The case was that of a female infant, aged 18 months, who was admitted to hospital in an unconscious state. Two months before admission the child had stopped talking and become dull and listless. For two weeks the legs had been stiff and crossed, and the pupils dilated. For the last three days the stiffness had increased, and opisthotonos had been noticed one day before admission.

On examination the child was quite unconscious, with half-open fixed eyes. The whole body was rigid, and the back arched with some head retraction. All four limbs were rigidly extended, and there was adductor spasm of the legs. All the deep reflexes were exaggerated ; the plantar reflexes were extensor and the abdominal absent. After lumbar puncture and the removal of 2½ oz. of clear colourless fluid under great pressure the rigidity diminished gradually, and for one day the limbs were almost flaccid. The following day the spasticity returned and the fontanelle became very tense. From this time onwards until death, no less than twenty-eight days later, *the child lay in a state of extreme rigidity in the extended position, the feet in a position of marked plantar flexion, and the thighs adducted. The upper limbs were so rigid that at times they did not rest on the bed ; the forearms were persistently pronated so that the palms were turned outwards, and the fingers flexed over the thumb,* *which was held across the palm.* Actual opisthotonos was, however, only once present. The jaws were usually tightly clenched.

Mention may be made in passing of the extraordinary pyrexia in the case, reaching on one occasion 111° F. in the rectum, and 109° F. in the axilla.

Post-mortem, a solid rounded tumour was found situated in the mid-line in the region of the corpora quadrigemina (fig. 9). It did not involve the pons, cerebellum or optic thalami, but pressed forward into the aqueduct of Sylvius, causing a block, with resulting distension of the third and lateral ventricles. The tumour was apparently of a glio-sarcomatous type.

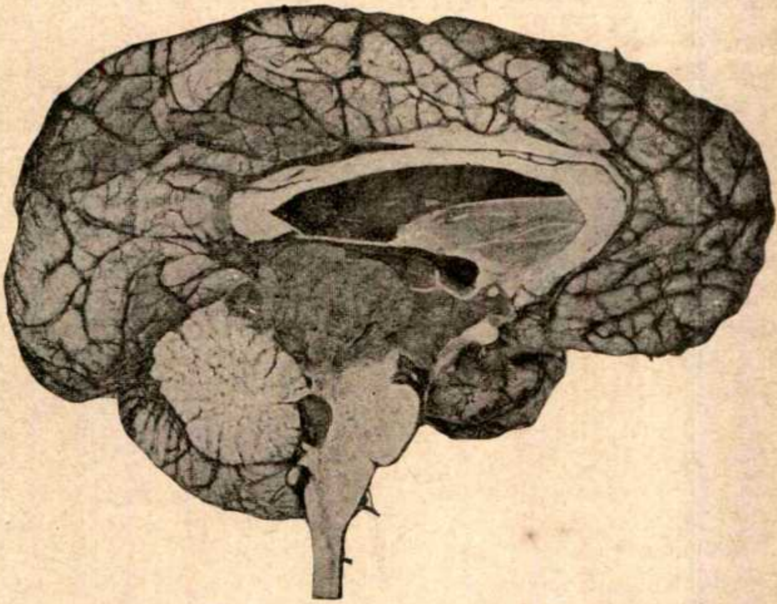


FIG. 9 (Turner's case).—The position of the tumour is such as to produce complete physiological decerebration at the level of the corpora quadrigemina.

These three cases in particular furnish ample corroboration of the connection between the peculiar symptomatology of decerebrate rigidity with postural fits and the neuronic level of the underlying lesions, and the identity of the postures in all confirms the support given by clinical observation to the findings of the physiological laboratory. The child in Turner's case was to all intents and purposes an experimental decerebrate preparation, and its survival for four weeks renders the case unique, as far as I have been able to discover, from this point of view.

It will doubtless occur to the reader that in various organic nervous conditions the phenomena under consideration make their appearance in greater or less detail. Thus, not a few cases of Little's disease conform strictly to the decerebrate type. Attention may also be directed to the cases published by Sutherland and Paterson [17] under the heading. "A Type of Cerebral Mal-development (Forebrain Aplasia)." In these infants imperfect development of the cerebrum with relative conservation of the neuraxis below the basal ganglia has led, as might be expected, to the appearance of the decerebrate attitude, as can be seen by a glance at the figures in the paper.

Further, the familiar head-retraction of various forms of basal meningitis, with or without actual opisthotonos, is certainly but a part of the complete decerebrate posture. Reference will be made to this point again.

II.—(1) CASES OF DECEREBRATE ATTITUDE IN CONSCIOUS LIFE.

There is no reason to doubt that the involuntary adoption, when consciousness is not in any way impaired, of an attitude corresponding exactly to the whole or any part of the decerebrate posture is to be attributed to the action of the same mechanism. The symptoms, it will readily be understood, are most apt to appear in circumstances under which the superior control of the corticospinal system is structurally or functionally defective; but, on the other hand, the characteristic postures are scarcely so likely to be maintained indefinitely as in the unconscious cases. There is, as it were, a conflict between two motor mechanisms, each attempting to jockey the other out of possession of the final common path.

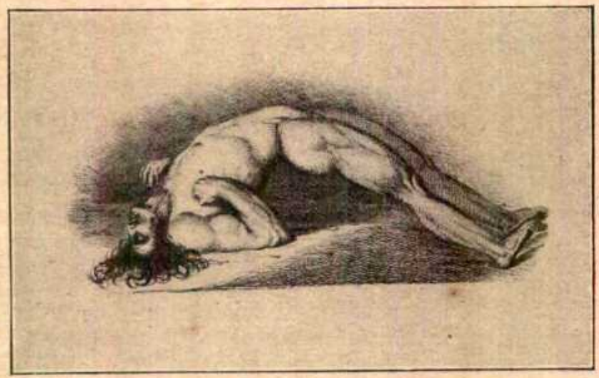


FIG 10.—Sir Charles Bell's drawing of a case of surgical tetanus.

I do not remember ever to have seen the assumption of the decerebrate posture in hysteria during consciousness, though a case of this description is figured by Dejerine in the second edition of his "Sémiologie" (fig. 228, p. 544). In cases of tetanus the tonic spasms, consciousness being unclouded, are often closely analogous to those we have been considering, as Hughlings Jackson emphasized—hence his expression "tetanus-like seizures" for tonic fits, already alluded to. Fig. 10 is a reproduction of Sir Charles Bell's drawing of a case of surgical tetanus, observed by him on the retreat with Sir John Moore to Corunna. The flexion of the arms, in contradistinction to the usual extension of the typical decerebrate position, has, of course, frequently been noted in "cerebellar fits," and its meaning is discussed below.

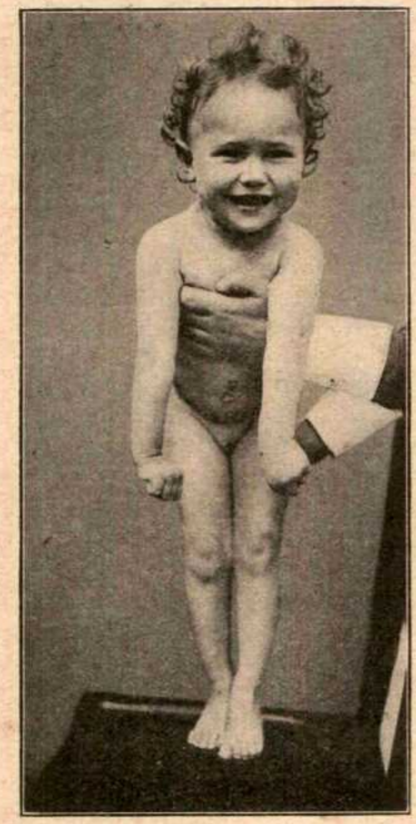


FIG. 11 (Case 9).—Decerebrate attitude of upper limbs in a case of diplegia.

Cases of more or less Complete Decerebrate Attitude.

Case 9.—E. W., aged 2, was admitted to the National Hospital under the care of Dr. Risien Russell on March 1, 1920.

The child was a nine months' infant, born naturally, and breast-fed.

Apparently no particular notice was taken of her condition till she reached the age of six months, when her inability to sit up was remarked on. Her mother, nevertheless, admitted on being questioned that the child "had never used the arms properly" and that the legs tended to be stiff at the knees. The child had never walked.

On examination no impairment of function of the cranial nerves was detected. Intelligence, as far as could be ascertained, seemed good enough; body and head were well-proportioned. With support the patient was occasionally able to take a step or two in a very imperfect way, getting on to her toes and lifting her feet high. There was considerable spasticity in the legs and some varying rigidity of the trunk. Much the most noteworthy feature of the case, however, was the attitude of the arms, one not persistently maintained, but adopted involuntarily and interrupted only by voluntary movements. When the infant was lifted up or supported in a more or less vertical position the limb attitude was especially manifest, and it was often maintained for many minutes at a time (fig. 11). The arms were adducted at the shoulders and sometimes rather advanced, fully extended at the elbows and powerfully inverted and hyperpronated, with wrists extended and hands closed in flexion, presenting an absolute identity with the attitude of decerebrate rigidity already described.

Case 10.—H.O., aged 9, had been born at full time in a normal confinement. She was always weak in her arms and legs, and had not walked till she was three, and then very imperfectly. The history given by the parents was that she was slowly getting worse and that for six weeks she had not been able to walk at all.

When she came under observation her intelligence was noted to be very poor, and examination was rendered unusually difficult owing to her inattention.

What struck the observer was the extreme degree of volitional imperfection as regards movement: the child seemed to be unable to move any limb volitionally at all, and to succeed in doing anything to order only, as it were, by accident. The musculature of the body and limbs was distinctly rigid on passive movement, and the hypertonus was more noticeable proximally than distally. The abdominal reflexes were absent and the plantars extensor.

At one time the patient seemed to lie in a position of more or less generalized flexion, and at another, of generalized extension. In the former of these the trunk was bent forwards and the chin almost reached the chest; the arms were adducted, elbows flexed, forearms pronated, crossing each other on the chest; the thighs were flexed and adducted and the legs flexed at the knees. This huddled-up attitude seemed to be assumed mainly, though not solely, during sleep. When the patient was disturbed, or her limbs handled dur[illegible] an examination, she almost always passed into an extensor attitude (fig. 12), with head retracted, arms fully extended, inverted and hyperpronated to an extreme degree: the legs were not, as a rule, fully extended at the hips or knees, though the feet were inverted, heels drawn up, and toes pointed down.

It should be mentioned that in addition to these attitudes the limbs were the seat of occasional slow spontaneous movements of an athetoid nature.

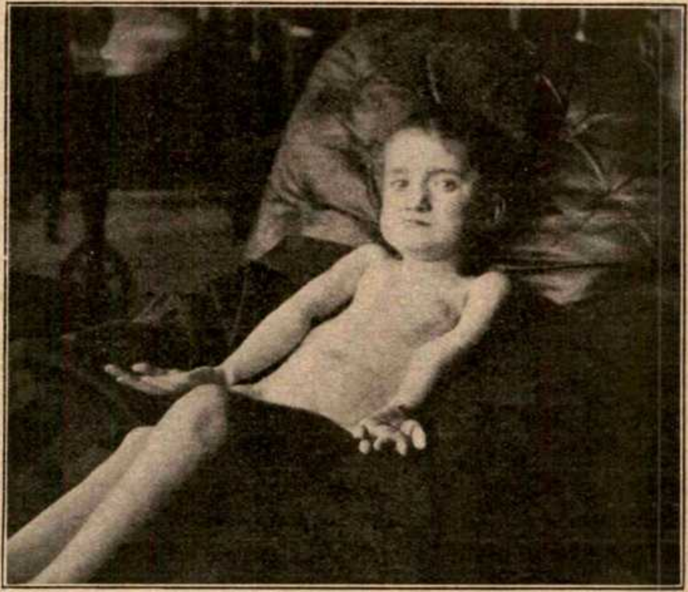


FIG. 12 (Case 10).—Extension-pronation attitude of upper limbs in a diplegic (see text).

(2) INCOMPLETE, LIMITED, OR FRAGMENTARY CASES.

Included in this section are cases where the decerebrate attitude is unilateral in arm and leg, or confined to one limb or segment of a limb. The more severe the degree of cortico-spinal interruption, corresponding to what obtains in actual decerebrate rigidity, the more likely is the unilateral attitude to resemble what is found in the complete cases, of which a number of instances have been furnished. It might be supposed, simply, that the ordinary hemiplegic attitude of arm and leg is a unilateral decerebrate posture, but this is the case only in a sense. One scarcely sees in experience the complete half attitude which would depend on the existence of complete unilateral cortico-spinal interruption, and this, owing to bilateral representation, cannot in fact r. The upper extremity, again, assumes an attitude in flexion at the elbow, commonly, and not extension; nevertheless, the adduction at shoulder, pronation of forearm, and flexion of wrist and perhaps fingers, are to be ascribed to the development, incomplete though it may be, of the decerebrate posture. It is important to recognize, in this connection, that in experimental unilateral decerebrate rigidity there are phenomena to be observed on *both* sides of the body. The following is Sherrington's [15] account of the condition:—

"If instead of both cerebral hemispheres one only, say the right, be ablated, the decerebrate rigidity appears, though not with the same certainty as after double ablation, chiefly on the same side as the hemisphere removed. The monkey, when slung after ablation of one—e.g., the right—hemisphere exhibits generally the following attitude. The right limbs are extended in the pose above described as characteristic for decerebrate rigidity, the tail is strongly incurvated toward the right, that is, its concavity is toward the right and its tip is also toward the right. It resists passive movement to the left, and if displaced thither immediately on being released flies back. The head also is pulled toward the right and retracted. The left forelimb . . . . is sometimes distinctly more flexed than would be expected in the paralysed condition of the animal; the left knee likewise. . . . . The contrast between the attitude of the crossed and homonymous sides is very striking . . . .

"Homonymous extensor rigidity consequent on ablation of one hemisphere is however neither so constant of production nor so persistent when it has appeared as the rigidity following bilateral ablation. After coming on it may totally subside and again reappear, and so on several times over. There would seem under these circumstances a struggle between two conflicting influences, as though a tonic influence from the still intact crossed hemisphere at times overcame and at times was overcome by another opposed influence from a lower centre. Some amount of extensor rigidity on the side opposite to the lesion is not uncommon."

Through the kindness of Dr. Poynton I am able to refer to a valuable case which he has afforded me every opportunity of examining, and which will serve to illustrate this part of our subject. The peculiar attitude presented by the child offers as close a resemblance to experimental unilateral decerebration as is likely to be noted in man.

Case II.—J. R., aged 2, was admitted to the Hospital for Sick Children, Great Ormond Street, on February 21, 1920, under the care of Dr. Poynton.

The mother's pregnancy was normal, and the child was born at full time by easy labour.

It was suspected in his second or third month of life he was not a normal child, because he was so quiet and inactive. He "lay like a doll" in bed or in his mother's arms. He was quite conscious, and took the breast readily, but moved neither legs nor arms, neither cried nor smiled, and apparently took no notice of his surroundings. About the age of 6 months he began to use his right arm, moving it about in a jerky way, and to draw up and kick his legs about. Since then there had been a gradual increase in the movements of the limbs, especially on the right. He had never been able to hold his head up, sit up, or stand, and had never learned to talk. During the last six months restless and jerky movements of the limbs became rather more obvious. There was no history of convulsions or fits at any time.

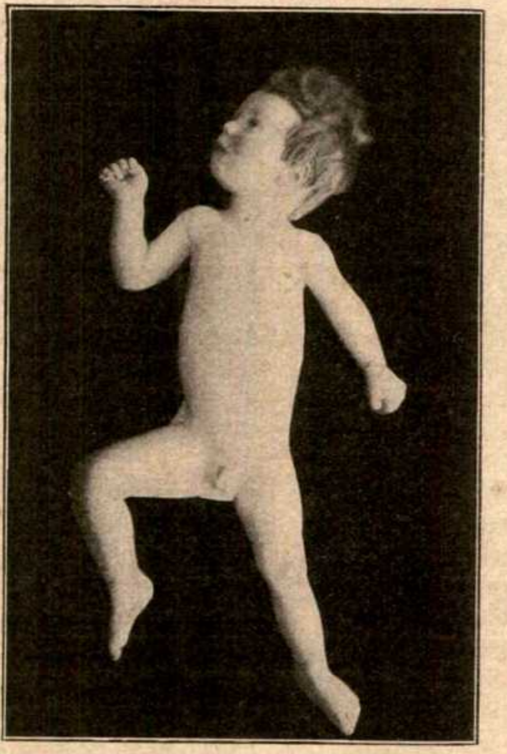


FIG. 13 (Case 11).—Attitude approximating to that of experimental unilateral decerebration. The photograph was taken from above, the child lying on his back on the floor.

On admission the child was seen to be a healthy-looking little boy, of almost normal weight and length for his age. He seemed intelligent and bright, and followed movements with his eyes, but did not understand the simplest spoken word. He cried if subjected to pain, and if hungry or interrupted during a meal, but did not smile or respond in any way to pleasurable stimuli, except to cease crying if he were already crying. He made no attempt to speak.

There was no true paralysis in any limbs, since the arms and legs could be moved in any direction, but their power was very feeble, and volitional control seemed extraordinarily erratic. The boy was unable to sit up, stand, or crawl, and if placed on his belly he could not turn over. There was a constant tendency to adopt a peculiar attitude (fig. 13); when lying on his back he practically always kept the right arm level with the shoulder in an abducted position, and flexed to a right angle at the elbow, the forearm midway between pronation and supination, the wrist somewhat over-extended and the fingers and thumb flexed; the left arm, on the contrary, was usually fully extended and close by the side or slightly abducted, forearm pronated and hand closed in a fist. The right leg was flexed at hip and knee, everted and abducted, with heel drawn up and toes pointing down; the left leg, on the other hand, was, like the homolateral upper limb, extended in full and sometimes slightly abducted. As a rule the head was turned to the right and somewhat retracted, chin tilted up and occiput lowered.

There was moderate tonus of the musculature; neither hypotonia nor rigidity or contractures were present.

In addition, however, frequent involuntary movements of a more or less choreiform character, jerky and apparently purposeless (objectively, at least), interrupted the maintenance of the posture above described. These were best seen when the child was held up by the axillæ, the limbs moving jerkily and erratically as in a Punch and Judy figure, the trunk wriggling and twisting and the head rolling restlessly.

Sensibility to pain appeared normal. All the deep reflexes were active; the abdominals were present and the plantars of the flexor variety.

The Wassermann test was negative in the blood and cerebrospinal fluid.

When we pass to what I am convinced are in fact one-limb or even unisegmental instances of the same decerebrate state the subject becomes still more interesting. Little, if any, attention has been paid to this aspect of the question, significant and informative though it is, although in 1872 Hughlings Jackson made a passing allusion to it in a few words: "In some cases of cerebellar disease there is only a fragment of the tetanus-like condition."

If we take, for example, the question of the head alone, neurologists are familiar with what has long been known as the "cerebellar attitude" of that part of the body. It is characterized, in unilateral lesions, by a tendency of the head to be tilted back and the occiput to be turned to the side of the lesion (fig. 14). To recall Sherrington's words in unilateral decerebrate rigidity: "The head is pulled towards the right (in right-sided lesions) and retracted." Now in complete or bilateral cases it is to be supposed that the *turning* elements to the right and the left respectively will neutralize each other, whereas the *retracting* element, common to both, will result in the accentuation of that posture. And this is precisely what is found in complete decerebration: the head is strongly retracted in the mesial plane, and rotation is absent. This is the common position in bilateral diffuse basal meningitis and other organic states. Should one (right or left) mechanism for some reason predominate over the other the lateral-tilting element will be reintroduced.

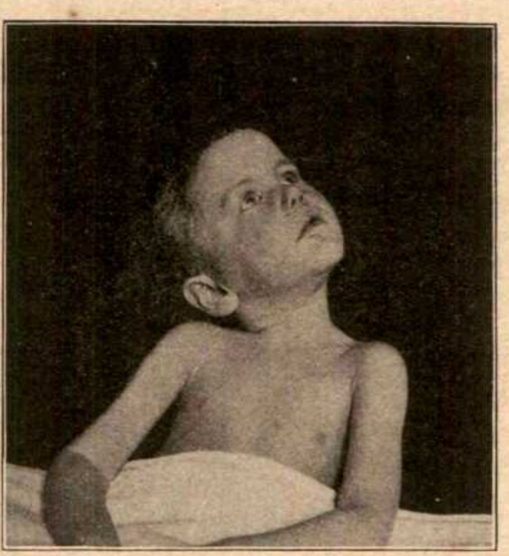


FIG. 14.—Illustrates the "cerebellar" attitude of the head in a case of right cerebellar tumour. The typical decerebrate attitude of the head is compounded of two partly opposing, partly reinforcing, "cerebellar" components.

A similar explanation holds good for curving of the trunk (spinal column). The opisthotonos of bilateral decerebrate rigidity is compact of right and left *extending* elements, reinforcing each other, whereas the right and the left *rotating* elements neutralize each other. Occasionally, however, in not quite complete cases, the lateral deviating element can be detected, as in the figures in Sutherland and Paterson's forebrain aplasia cases. It will be remembered, further, that in the tonic fits of Case 7 lateral-rotating mechanisms were in full activity during part of the seizures. Probably there is a similar dissociation of "corresponding opposites" in the phenomenon of skew deviation.

Though, as remarked above, such head and other attitudes are commonly spoken of as "cerebellar," it has been shown that they are in reality part and parcel of the postures of decerebration; hence the term "cerebellar" is doubly misleading, since it fails to denote this integral association, and since, as will be shown, it is doubtful whether the origin of the attitudes is cerebellar at all. Clinically, in not a few cases of head deviation that I have seen, the lesion has been mesencephalic and the cerebellum quite intact.

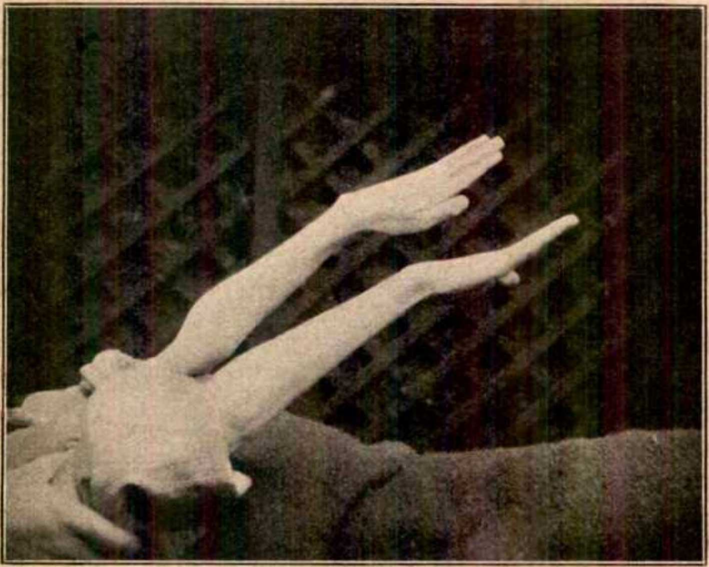


FIG. 15 (Case 12).—A fragment of decerebrate posture. The left arm shows the pronation phenomenon in a case of tumour of the left edge of the middle lobe of the cerebellum.

To pass, in the next place, to instances of the condition being confined almost to a segment of a limb, or even to one phase of segmental movement, particular attention may be directed to the "pronator sign," which is an important part of the complete decerebrate posture. Nothing is more characteristic than this pronation or overpronation; it was noted by Sherrington in his experimental animals, and we have seen the frequency with which it occurs in human cases of cortical "ablation." In certain cases of limited lesion dissociating cortico-spinal and mesencephalo-spinal function unilateral pronation of the forearm has been the sole definite and persisting sign, a small but integral fragment of the complete decerebrate attitude, and therefore of considerable clinical significance.

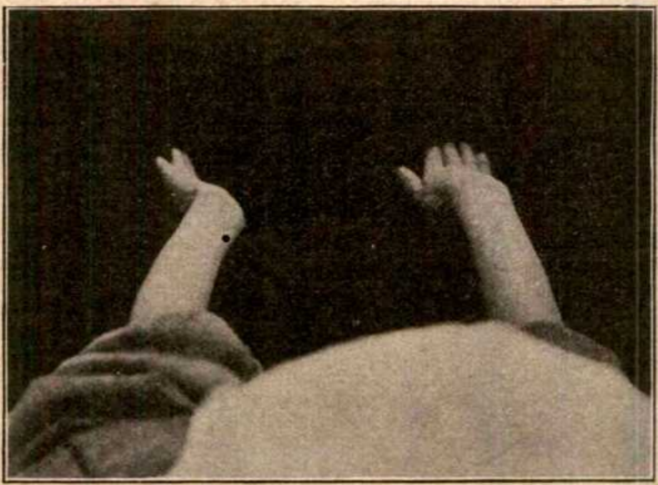


FIG. 16 (Case 12).—Photograph taken from behind the patient's head. The pronator sign in the left arm is an integral part of decerebrate posture.

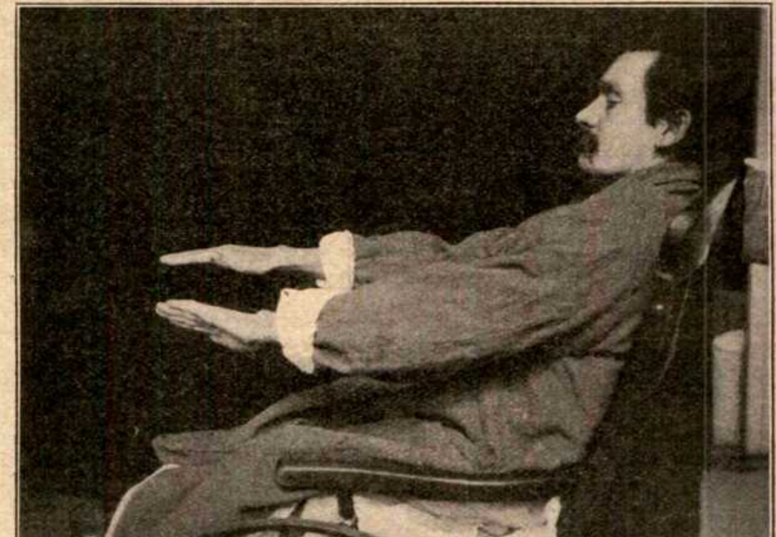


FIG. 17.—Pronation of the left forearm in a case of cerebellar disease, presumably in the left of the middle obe.

Case 12.—E. L., aged 12, had suffered for one year from the usual symptoms of intracranial tumour, viz., headache, giddiness, vomiting, and was found on examination to have well-marked double optic neuritis. Almost the sole localizing sign was a somewhat indefinite reeling gait, pointing, however, on the whole, to a lesion in the left cerebellum. Accordingly, the patient was trephined over this area. A great part of the left lateral lobe of the cerebellum was removed under the impression that it was gliomatous, but eventually a quite small encapsuled tumour, about the size of a cherry, was discovered at the left edge of the middle lobe. During the operation pronounced skew-deviation made its appearance, the left eye being down and in, and the right eye up and out. When the patient was being tested some ten days after the operation it was noticed that the left arm, when extended, always assumed an attitude (figs. 15, 16) of marked pronation and inversion—a symptom which had not been present before, and in addition the left leg was held extended and inverted at the foot. Since the operation, further, the head definitely assumed the posture of occiput to the left, chin to the right. A month later these postural attitudes were still maintained.

Fig. 17 is from a case of cerebellar disease exhibiting the same phenomenon as in Case 12. The presumption is that the lesion was in an identical spot, viz., the left side of the middle lobe; no opportunity of corroborating this, however, was afforded.

(3) CASES OF PARTIAL DECEREBRATE ATTITUDE OCCURRING AS INVOLUNTARY MOVEMENTS IN THE COURSE OF CHOREA AND ATHETOSIS.

Finally, I have for a long time been impressed with the way in which this pronator sign, a fragment of decerebrate posture, shows itself in cases of involuntary movement, more particularly in chorea and athetosis.

When the patient with acute or subacute Sydenham's chorea is asked to extend his arms above his head, palms to the front, it is extremely common to find them adopting involuntarily an attitude of pronation, with the palms turned outwards. The movement is well seen by contrast in cases of hemichorea (fig. 18), and is sometimes found persisting after the actual choreic movements have ceased. It is also to be observed in so-called paralytic chorea. Thus through the changing foreground of choreic twitching can be recognized a postural background of mesencephalic significance.

Similarly, in athetosis the pronator attitude is a position of election; it is, for example, vastly more frequent than any attitude of supination. Difficult though it be to follow the changing movements of athetosis,

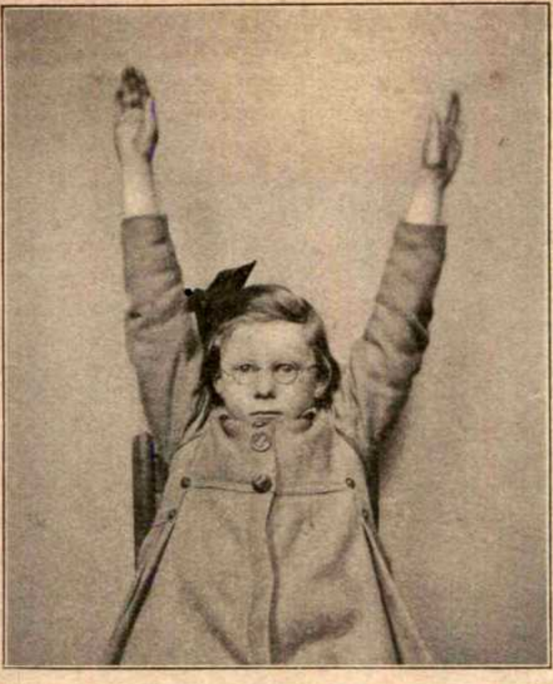


FIG. 18.—Well marked pronator sign in a case of left unilateral Sydenham's chorea (hemichorea).

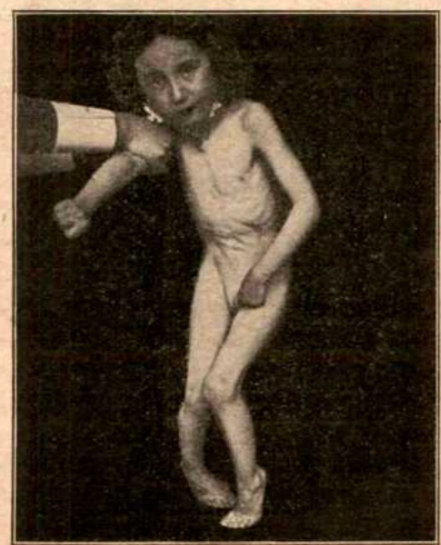


FIG. 19.—Characteristic transient decerebrate posture of right upper limb in a case of diplegia with athetosis.

there is, in my opinion, no doubt that the substantial background of that condition is one of varying decerebrate posture. Only one case need be cited in support of this contention; others are sufficiently illustrated in the photographs (figs. 19, 20).

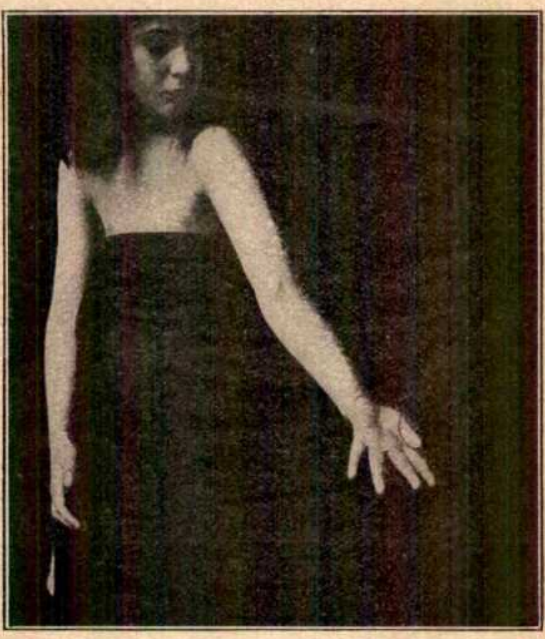


FIG. 20.—Case of infantile cerebral hemiplegia with athetosis (left). Extreme hyperpronation and characteristic decerebrate posture, with the exception that the fingers are extended and not flexed.

Case 13.—A. W., aged 10, developed diphtheria in January, 1919. The attack was of moderate severity. About fourteen days after the commencement of the illness, and while the patient was still very ill, she developed a complete left hemiplegia, involving face, arm and leg, without loss of consciousness and without any fit. Some weeks after the attack it was observed that the left arm was the seat of slow involuntary movements of an athetoid nature.

When the patient came under observation in June 1919, the hemiplegia was noted to be slight in degree, implicating mainly the left face and the left arm. Though the leg was not much complained of an extensor response was present. The left arm showed fair muscular power, though weaker than the right, and a fair range of voluntary movements, but these were interrupted by characteristic involuntary athetoid movements, more especially of the forearm, hand and fingers. There was a constant tendency for these movements to place the forearm in the attitude of over-pronation (fig. 21). Under treatment steady improvement took place, till at present the athetosis has very largely disappeared; but the pronation-posture, though less frequently assumed, is still to be observed, especially if the patient is asked to extend her arms above her head.

In addition to the pronator sign it is possible to pick out others of the postural manifestations during the maintenance of decerebrate rigidity and observe their reproduction in the course of chorea and athetosis. One more phenomenon may be alluded to in this respect. It will have been noticed that in the great majority of the complete cases the arms are flexed at the wrists; Sherrington's experimental animals showed the same flexion. Such wrist-flexion is, of course, the usual attitude at that joint in hemiplegia, representing, as has already been said, a fragment of decerebrate posture, and precisely the same flexion is extremely common in the choreic hand (fig. 22); it is always well seen when the arms are voluntarily extended in front. The choreic hand has long been known, but its significance in connection with decerebration does not appear to have been observed. Coupled with the flexion of the wrists is a tendency to finger over-

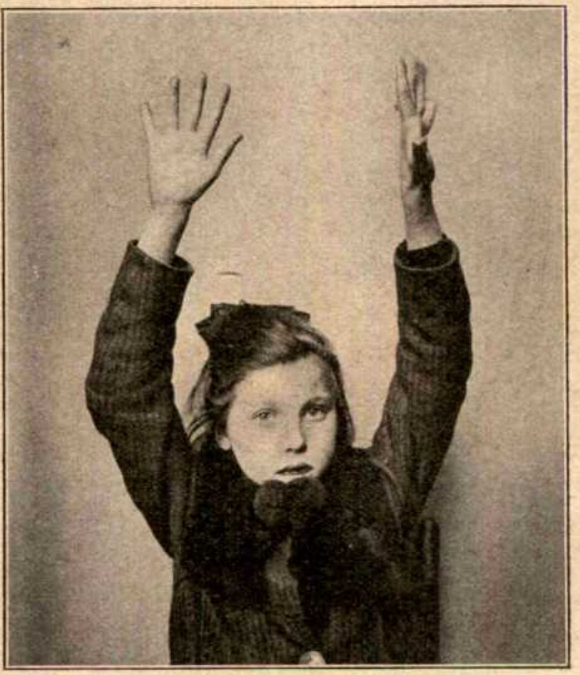


FIG. 21 (Case 13).—Slight left hemiplegia with athetosis, following diphtheria. The left arm adopts an involuntary position of over-pronation.

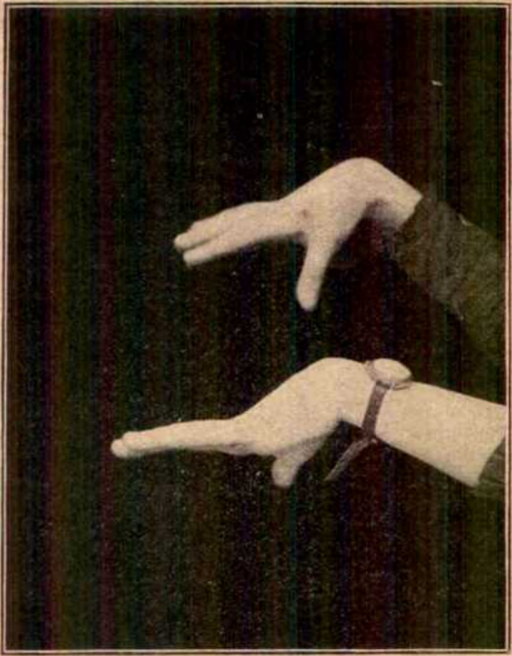


FIG. 22.—The choreic hand ; wrist flexion and some over-extension at the knuckles.

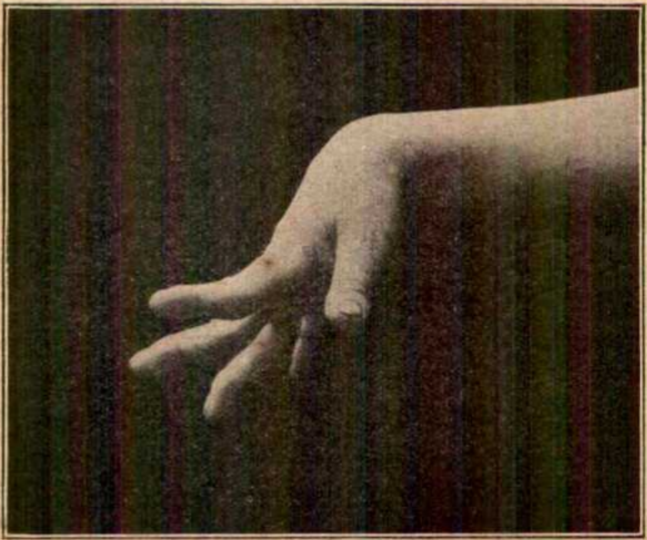


FIG. 23.—The athetoid hand, practically identical with the choreic hand (fig. 22).

extension, which is not observed generally in decerebrate rigidity (though in Case 8 the fingers were extended and adducted), but which, it must be understood, is largely voluntary in the test as usually employed, the patient being asked to "hold his hands out in front." This same flexion of the wrists is one of the commonest attitudes of athetosis (fig. 23).

There has recently been under my care at the National Hospital a case in which this combined wrist-flexion and finger-extension was a constant phenomenon in a child whose physiological condition was one not far from complete decerebration, and in whom the vestibular reflexes of Magnus and De Kleijn [14] were readily obtainable.

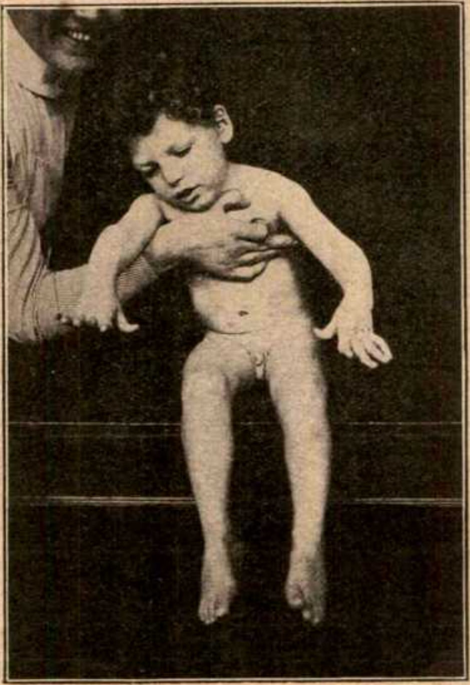


FIG. 24 (Case 14).—Microcephaly with diplegia and athetoid attitudes. For significance see text.

Case 14.—R. G., aged 3½, was admitted to the National Hospital under my care on June 5, 1919.

The infant was born after long labour with instrumental delivery. He was always backward, never tried to crawl, and could not use his arms properly. He never could hold his head up as other children.

On admission the child was noted to have a somewhat microcephalic head. He was extremely backward and infantile, though well enough developed physically. Little if any attention seemed to be paid to interest taken in him. He wailed in a monotonous voice most of the time when left alone, but usually stopped when addressed, and occasionally an infantile smile crossed his features. The more or less constant attitude of the child is seen in fig. 24.

Magnus and De Kleijn reflexes.—When the child was placed on its back and the head gently turned, say to the left, and when it stopped crying, it lay quietly with upper limbs in a moderate flexion posture. On turning its head sharply to the right passively an immediate extension of the left arm into the usual decerebrate attitude was always obtained.

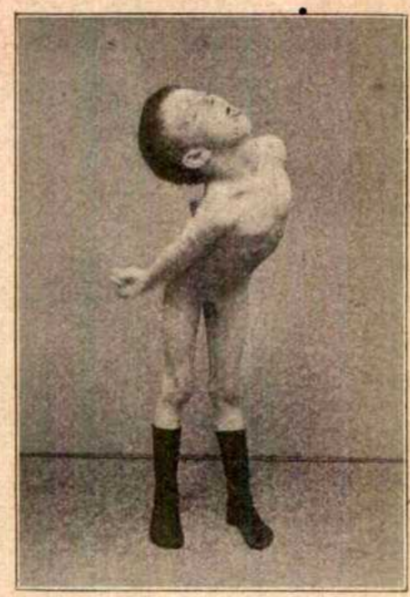


FIG. 25.—Torsion-spasm. The right arm is in a characteristic decerebrate posture.

One other variety of involuntary movement may be alluded to. In the rare disease known as torsion-spasm or torsion-dystonia (dystonia muscularum deformans) one or other limbs may adopt the decerebrate posture in its entirety. A striking case of this description under the care of my colleague Dr. Collier, who has given me every opportunity of examining it, has been fully reported by Dr. M. A. Blandy [2] and a photograph of the boy is here reproduced (fig. 25).

To recapitulate, thus far, in a sentence or two, the general view taken is as follows :—

(1) The symptoms of acute cases of physiological decerebration in men are identical with what obtains in animal experimentation. The decerebrate condition of forearm, wrist and fingers—extension at elbow,

pronation, flexion of wrist and usually of fingers—is commonly more marked than in animals, sometimes to an extreme degree. It is particularly noteworthy that respiratory disturbance often precedes or accompanies the decerebrate rigidity. The condition is frequently combined with tonic or postural fits, the feature of which is that they accentuate or reinforce the existing decerebrate attitude.

(2) Tonic or postural fits are, in reality, the transient adoption of this decerebrate attitude, complete or incomplete as the case may be. In the case of the fits there are changing phases of tonic attitude or posture which are less likely to be observed in the greater fixity of decerebrate posture produced by the release of certain neural mechanisms in structural nervous disease which *permanently* dissociate cortex from mesencephalon.

(3) The complete decerebrate attitude is compounded of unilateral elements which reinforce each other and of other unilateral elements which neutralize or antagonize each other. In the case of head and trunk postures, at least, it is possible to resolve them into components which, in incomplete cases, may preponderate on one or other side, producing deviations from the complete posture. This complete decerebrate posture is essentially composed of movements grouped in action in the mesial plane of the body.

(4) In cases where consciousness is intact and the corticospinal system partially or entirely in functional abeyance, portions of the decerebrate attitude make their appearance in the form of head, trunk, or limb deviations and attitudes; there may be fragments confined to one limb or segment of a limb. This is in particular the case with the pronator sign.

(5) In involuntary movements of the nature of chorea and athetosis it can be shown that there is a postural background which is composed of part of the complete decerebrate attitude. In both of these varieties the commonest phases are pronation and wrist flexion; the choreic and the athetoid hands are identical in this respect, and pronation and wrist flexion are an integral part of decerebrate posture.

(6) There is an important difference between the unconscious and the conscious cases, seeing that in the latter, unless the corticospinal interruption is absolute, the cortex may and often does interfere in part with the activity of the released lower mechanisms. In Sherrington's words, " there is a struggle between two conflicting influences."

THE PATHOGENESIS OF DECEREBRATE RIGIDITY.

We have seen that the resemblance between human and animal decerebrate rigidity is of the closest, and pathological evidence has been adduced indicating the nature and localization of the morbid processes likely to bring it about. From the type of pathological process, however, herein described as producing the syndrome (tumours, cerebral hæmorrhage, meningitis and internal hydrocephalus), it is apparent the localization can only be approximate, and physiological rather than anatomical; with other words, any lesion dissociating the cortex from the mesencephalo-cerebellar level and at the same time leaving the latter functionally intact, is calculated to cause the rigidity to develop, but the finer anatomical details can only be supplied at present from the experimental side. At the same time attention may again be directed to the fact that in the acute human cases marked disturbance of respiration has been repeatedly observed (see the cases in the first group, above). Graham Brown [10] has shown by exact experimentation that there is a respiratory tract, localizable with extreme nicety, in the mid-brain. In the ape it lies about 3 mm. from the aqueduct, and its stimulation produces gasping or panting movements. The occurrence of respiratory difficulty in the acute human cases may, perhaps, be attributed to interference with the function of this tract (assuming its presence in man) and thus provides important collateral evidence suggesting that the vicinity of the nucleus ruber (as is shown below), and not the cerebellum, is the neighbourhood whence is derived the activity that produces decerebrate rigidity.

The general physiological view enunciated by Hughlings Jackson [12] in 1877 as to the cause of hemiplegic rigidity is the starting point in our investigations of the pathogenesis of the symptom-complex under consideration. The following is his outline of the basic features of the "co-operation of antagonism" between cerebrum and cerebellum.

"There is evidently a duplex symptomatic condition, negative and positive; with loss of power over the muscles there is tonic action of them. Negative states of the nervous centres cannot *cause* positive states of muscles, they may *permit* them. My speculation is that the rigidity is owing to unantagonized influence of the cerebellum. . . . The cerebellum is the centre for continuous movements and the cerebrum for changing movements. . . . The former are tonic, the latter clonic. . . . In walking, the cerebellum preserves the equilibrium of the body, tends to "stiffen" all the muscles; the changing movements of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. . . .

"When, however, the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia, from the parts which it most specially governs, the cerebellar influence is no longer antagonized; there is unimpeded cerebellar influx, and hence rigidity of the muscles which in health the cerebrum chiefly innervates. The principle, I think, accounts for the duplex, negative and positive conditions in the contractures of hemiplegic children, in *the athetosis of Hammond*, and accounts for the rigidity and "propulsive" walk in paralysis agitans; temporary removal also for tetany and *certain hysterical attacks*, and also for some post-epileptic conditions of movements" (italics mine).

Specially noteworthy in this account is Jackson's recognition of "cerebellar influx" both in athetosis and in certain hysterical attacks; with characteristic prescience he sketched a hypothesis half a century ago, many of the details of which are only now receiving complete confirmation.

In regard to the above theory, however, the chief comment to be made is that the speculation as to the actual site of origin of the rigidity-producing influence being in the cerebellum cannot be considered proved, though the latter organ plays an important part. There is no question that the phenomena of decerebrate rigidity are release-phenomena, and that, again in Jackson's words, a lower influx invades what the higher gradually abandons.

For the determination of further essential facts in connexion with the subject of pathogenesis we must turn to the work of the experimentalists, conflicting though it is in some respects and not always of simplicity in interpretation.

In the first place, there is general agreement that decerebrate rigidity makes its appearance after transection of the neuraxis at the level of the anterior colliculi, and disappears by a second section below the neuronic level of the medulla. For its development, therefore, we are limited to the mesencephalon, cerebellum, and pons. It must, nevertheless, be mentioned there is experimental evidence suggesting that similar attitudes can be obtained when the decerebration is higher, leaving intact the basal ganglia, in particular the optic thalamus, and that movements analogous to those of tonic fits can be produced by stimulation of the latter structure (Lowenthal and Horsley [13], Thiele [18], Graham Brown [8]). Rigidity, however, sets in with precision from mesencephalic transection, and for the moment we will confine consideration to the three parts of the neuraxis just specified.

This mesencephalic decerebrate rigidity, once established, can be abolished by various procedures. It will be readily understood that integrity of the lowest tone-effecting spinal proprioceptive arcs is assumed in this connection; with rigidity at its height, section of afferent roots at once abolishes rigidity for the limb concerned. Ordinarily speaking, therefore, for rigidity to appear a normal neuraxis below the mesencephalic level is presupposed, and if, clinically, a hæmorrhage spreads down the cord to invade local proprioceptive arcs the phenomena are certain to vanish. Thus the attitude of decerebrate rigidity may only be a phase in the course of a case and may disappear *sub finem*. Graham Brown [7], however, has made the important observation that integrity of these lower tone-effecting arcs is *not* absolutely essential. " The reactions (of decerebrate attitude) may occur many months after division of the dorsal spinal roots of the arm. That is to say, appropriate stimulation in the region of the midbrain may evoke an extensor postural tonus or a flexor postural tonus. Sherrington has found that the decerebrate rigidity which occurs after removal of the cerebrum does not occur in a ' de-afferented ' limb, but the fact that a condition which at any rate very closely resembles this state may be evoked in such limbs seems to point to the conclusion that the absence of this postural tonus in the decerebrate ' de-afferented ' animal is due to the failure of the ascending impulses from the limb which normally play—however indirectly—upon these mechanisms of the midbrain, and that the mechanisms themselves if properly activated are still able to induce the tonus."

In addition to the local spinal reflex arcs for tone, it is held by Sherrington, as Graham Brown says in the above citation, that impulses ascend to the midbrain mechanisms to maintain them in activity, but they do not pass by the dorsal columns, for section of these has no effect, whereas section of the ventro-lateral cord abolishes the rigidity. This suggests the conveyance of maintenance-impulses by the spino-cerebellar tracts, and Weed [20] believes the path is the ventral spinocerebellar tract of Gowers, and not the dorsal, since section of the inferior cerebellar peduncles, by which the dorsal pass to the cerebellum, is without influence on established rigidity.

Another method of abolishing the extensor tonus after decerebration is by excitation of the anterior (superior) surface of the cerebellum, from near the midline outwards to near the lateral border of the cerebellar surface (superior vermis and outward). This has been observed repeatedly by Sherrington, Lowenthal and Horsley, Weed and others. If, then, stimulation of this part of the cerebellum inhibits an induced decerebrate rigidity, it might be supposed, conversely, that destruction of the same area would produce the condition, mesencephalic decerebration not being performed, and this has been done by Sherrington and by Thiele. Bisection of or injury to the vermis, median section of the cerebellum, and partial ablation of the cerebellum, are all apt to produce the phenomena under discussion, but Sherrington is not convinced that the two conditions are identical, and specially states that after these cerebellar operations decerebrate rigidity "sets in often, *but not always*" (italics mine). He has seen the condition "persist after removal of the cerebellum, if the latter ablation be performed without any serious amount of hæmorrhage." Graham Brown, similarly, has clearly shown that the reactions of decerebrate rigidity may be obtained from stimulation of the midbrain "after the complete removal of the cerebellum"; and Thiele has removed the whole cerebellum down to the paracerebellar nuclei (Deiters) without affecting the decerebrate posture.

It is apparent, therefore, that functional integrity of the cerebellum is *not* a *conditio sine quá non* for the appearance of the syndrome after mesencephalic transection in animals, and that the phenomena may develop in the actual absence of that organ.

These experimental findings seem to render an actual cerebellar origin for the rigidities improbable.

The position reached by Sherrington, Weed, and Graham Brown is that the mesencephalon itself is of prime importance. According to Weed, the real origin of decerebrate rigidity is in the midbrain in the region of the colliculi. "In this portion of the midbrain, lying ventral to the colliculi, the tegmental structure which apparently affords origin for the rigidities is the nucleus ruber." Graham Brown has developed the hypothesis in greater detail and with a wealth of experimental material. He has shown that "the red nucleus is the centre the activity of which conditions" the postural reflexes constituting decerebrate rigidity. And in a personal communication to the writer Professor Sherrington says:—

"The neighbourhood of the iter taken broadly so as to include the red nucleus, small-celled and large-celled parts, certainly has large postural (tonic) functions, and Graham Brown's experiments observed those in detail. Work on decerebrate rigidity has gradually come to suggest that the red nucleus region is of capital importance for the general reflex of 'standing' ('standing' in its various forms and sub-postures). Tonic (posture) fits would, to judge from laboratory observations, be likely to result from lesions of an acute kind in that region."

From the point of view of experimentation, then, the conclusion seems clear enough that the withdrawal of cortical control, by decerebration, from the spinal motor centres allows them to be played on by a non-pyramidal motor influence of a tonic nature, or rather allows this doubtlessly constant tonizing factor to reveal itself in action. The centre of this attitude- or posture-producing motor activity is apparently the nucleus ruber of the mesencephalon, and not the cerebellar cortex or cerebellar nuclei, and it is maintained in tone itself, ordinarily, by afferent impulses reaching it, "however indirectly," from lower (spinal) neural levels, though its activities are not necessarily paralysed by their absence.

The nucleus ruber, however, is no isolated and autonomous ganglion, but has numerous and important connections with structures above, beside, and below it. Further, and of equal importance, it is a twin nucleus. We cannot suppose, though it be the originator of decerebrate rigidity, that it is uninfluenced by stimuli from parts with which it is in close relation.

(1) Though the postural discharges of decerebrate attitude have not their origin in the cerebellum, the latter must certainly play a part, as long, that is, as it is in functional connection with the midbrain. We have already seen that the rigidity can be inhibited by excitation of the neighbourhood of the vermis, while lesions there would appear themselves to be provocative of rigidity, the mesencephalon being intact, or to accentuate existing rigidity. This experimental finding is supported by the clinical evidence of Case 12 above, since the destructive lesion allowing the "fragments" of decerebrate posture (pronation) to appear was situated at the left edge of the middle lobe, i.e., in the position whence decerebrate rigidity can be produced experimentally by destructive lesions. Thus the cerebellum would appear to exercise both an inhibitory and an excitatory action on the mesencephalic mechanism which is responsible for decerebrate posture. According to Weed, who produced the condition by removal of the cerebral hemispheres, leaving the internal capsules and basal ganglia (if I understand his descriptions correctly), it is possible to trace an inhibitory pathway from the cerebrum which corresponds to the fronto-

ponto-cerebellar route (viâ the middle cerebellar peduncle). Stimulation of this pathway always inhibited existing decerebrate posture, mainly homolaterally, suggesting a doubly crossed connection. Weed found the path is distributed, in part at least, to the cortex of the anterior portion of the superior vermis, whence, as has been said, previous experimenters have been able to inhibit the rigidity, but whether the inhibitory effect is exercised upon the basal cerebellar nuclei (Deiters) or upon the mesencephalic nuclei is not at present known.

As far, then, as cerebellar action is concerned, it may be tentatively concluded that (1) decerebrate attitude of mesencephalic origin is maintained by afferents reaching its source by spinocerebellar paths, and (2) inhibited by stimulation of a ponto-cerebellar path viâ the neighbourhood of the vermis, i.e., of what constitutes largely the palæocerebellum. Possibly the maintaining influence reaches the twin red nuclei by the superior cerebellar peduncles, since their section, in the decerebrate preparation, is usually followed by disappearance of the rigidity; as already noted, it is not certain how the inhibiting influence is conveyed to the mesencephalon, or even whether it may not pass back again to spinal centres by another route.

(2) The basal ganglia have intimate connections with the red nucleus. In particular, the corpus striatum is in relation with it by means of the ansa lenticularis. As our subject is mainly concerned with the phenomena appearing after transection through the midbrain, when the basal ganglia are out of action, it is not necessary to discuss the physiological connections of the two in this place, though several allusions have already been made to the fact that with suitable experimentation the rubral mechanism of posture can be activated from above, from the basal ganglia. In a subsequent communication concerned with the pathogenesis of involuntary movements further opportunity will be taken to analyse the bearing of the influence of the basal ganglia on mesencephalic function, and a brief reference to the connection of the red nucleus with tremor is given below.

We may turn next to the question of the rigidity-producing mechanism itself.

The careful work of Graham Brown has shown that in the decerebrate preparation electrical stimulation of certain points in the cross-section of the midbrain at the level of the anterior colliculi in monkeys yields characteristic reactions. The areas excited, his "focal points," lie on either side of the midline some millimetres ventral to the aqueduct of Sylvius, and correspond with the anatomical position of the red nucleus (and possibly, also, with that of the posterior longitudinal bundle). "Stimulation applied to this area of the cross-section of the midbrain on one side evokes a state of flexion in the arm of the same side and a state of extension in the arm of the opposite side. On cessation of stimulation these reactions are continued as maintained postures. The movements of the arms are accompanied by movements of other parts of the body. The tail seems to bend to the same side; the lower limb of the same side extends while that of the opposite side flexes; the head is rotated in such a manner that the face is turned away from the side stimulated." He has shown, further, the mutual antagonism of the right and left focal points, and by immediate or successive compounding of the two has produced bilateral extension, bilateral flexion, and so on. "With varying values of stimulation varying values of contralateral extension and ipsilateral flexion might compound to give reactions in which extension and flexion exhibited differing preponderancies."

Now our clinical study has already demonstrated the fact that the clinical phenomena of decerebrate rigidity are compounded of elements some of which are mutually reinforcing and others mutually antagonistic, and in Graham Brown's work we have experimental proof of the way in which these symptoms are produced. It is evident that the varieties and alternations of positions—e.g., all limbs extended, as is usual in human decerebrate rigidity, or arms flexed and legs extended as in Jackson's "tetanus-like seizures"—are due to compounding of varying influences affecting the red nuclei or originating in them. And it seems no long step to the supposition that unisegmental attitude, as, for instance, in the pronator sign, is the result of limited or partial red-nuclear activity.

It may be suggested that in the red nucleus, perhaps in the pars magnocellularis, are represented combinations of the ventral cell-groups of the spinal cord, linked up according to their flexor, extensor, pronator, supinator, &c., functions. For we cannot believe that in the red nucleus we have other than a "re-presentation" of the ventral cord cell groups. Graham Brown has, indeed, demonstrated that the point of common antagonism between the twin nuclei is below the midbrain, and the natural surmise is that it lies at as low a level as that of the spinal centres. If this be the case, then stimulation of a point in the red nucleus, after decerebration, may be supposed to innervate a physiological group of ventral cells, the flexors, say, scattered throughout the various spinal segments, and all linked to a nodal cellular representative in the former nucleus. The same must be true of extensor cells, &c. Uncontrolled activity of the twin nuclei from decerebration throws the individual into the decerebrate posture, with predominance or changing activity of flexor or extensor attitude on one or other side, in one or other limb, as the case may be, owing to changing influences in the red nuclei or reaching them.

Undoubtedly the predominating activity of the uncontrolled nuclei is one causing extensor tonus. The "neural balance" of a limb-pair, taken together, seems for some reason to be dominantly weighted or disposed towards, or "set in," extension [9]. Sherrington's explanation of this is that decerebrate posture is in reality "reflex standing," hence the preponderance of extensor tone.

Throughout this discussion, for the sake of clearness, the assumption has to some extent been made that it is precisely the red nucleus in which these postural activities are anatomically centred, though greater certainty is necessary before the matter can be regarded as settled. But it seems desirable to point out that the nucleus ruber is but a part of Edinger's nucleus motorius tegmenti, which consists of associated motor-cell groups scattered through the pons and mesencephalon to the level of the ventral optic thalamus, including Deiters' nucleus, and that the effect of stimulation is not likely to be limited to one descending rubral path, viz., the rubrospinal. There is, in fact, doubt as to the path or paths by which rubral activities reach the cell-groups of the ventral cornua; further, rubro-pontine and rubro-bulbar connections must not be ignored. In this connection it is well to mention that Thiele [18] attributes the conduction of decerebrate impulses spinalwards, not to the nucleus ruber and its connections, but to Deiters' nucleus, another part of the nucleus motorius tegmenti, and to the vestibulospinal tract. Thus there is room for further research and elucidation in the matter. It is clear, for example, that other influences reach the final common path of the lower motor neuron than the rubrospinal. The possibility of labyrinthine agency contributing a quota to decerebrate rigidity, originally mooted by Ewald, has been largely supported recently by the researches of Magnus and de Kleijn [14], who have proved that in the decerebrate animal passive movements of the head give rise to alterations of tonus in the limbs, and that each movement of the head varies in its influence on tonus according to the position of the animal in space. Only this passing allusion to their investigations can be now made.

Although the question is one to be dealt with elsewhere, brief mention may be made, in conclusion, of the connection of the red nucleus with involuntary movement. If the hypothesis suggested above be tenable, viz., that the flexor, extensor, &c., groups of the spinal cells are respectively linked to nodal representatives in the nucleus—are, as it were, re-assembled in these twin ganglia—then we can understand how tremor may occur in association with rubral disorder. For, in Jackson's words once more, "tremor differs from rigidity, not fundamentally, but in degree."

REFERENCES.

[1] BASTIAN. "A Case of Apoplexy in a Boy aged 15; Intraventricular Hæmorrhage, Convulsions, and Death in four hours," *Trans. Clin. Soc.*, 1884, vol. xvii, p. 21.

[2] BLANDY. "A Case of Torsion-dystonia, or Torsion-spasm," *Journ. of Neur. and Psych.*, 1920, vol. i, p. 148.

[3] BRUCE and DRUMMOND. "A Case of Intracranial Aneurism in a Young Subject," *Rev. of Neur. and Psych.*, 1904, vol. ii, p. 737.

[4] BRUCE, PIRIE, and MACDONALD. "Aneurism of the Anterior Cerebral Artery, with unusual Prolongation of Life after Rupture; Autopsy," *Rev. of Neur. and Psych.*, 1908, vol. vi, p. 449.

[5] ESKRIDGE. "Tumour and Large Cyst of the Cerebellum, with Symptoms extending over several Years," *Med. Record*, August 17, 1895.

[6] FIRTH. "Cerebellar Fits of the Hughlings Jackson type," *Journ. of Neur. and Psych.*, 1920, vol. i, p. 48.

[7] GRAHAM BROWN. "On Postural and Non-postural Activities of the Mid-brain," *Proc. Roy. Soc.*, B, 1913, vol. lxxxvii, p. 145.

[8] *Idem.* "On the Effect of Artificial Stimulation of the Red Nucleus in the Anthropoid Ape," *Journ. of Physiol.*, 1915, vol. xlix, p. 185.

[9] *Idem.* "The Rule of Reflex Response in the Limb Reflexes of the Mammal and its exceptions," *Journ. of Physiol.*, 1912, vol. xliv, p. 125.

[10] *Idem.* "A Respiratory Tract in the Mid-brain," *Proc. Physiol. Soc.*, May 16, 1914.

[11] HUGHLINGS JACKSON. "Case of Tumour of the Middle Lobe of the Cerebellum: Cerebellar Paralysis with Rigidity (cerebellar attitude); occasional Tetanus-like Seizures," *Brain*, 1906, vol. xxix, p. 425.

[12] *Idem. Med. Examiner*, 1877, April 5, p. 271.

[13] LOWENTHAL and HORSLEY. "On the Relation between the Cerebellar and other Centres, &c.," *Proc. Roy. Soc.*, 1897, lxi, p. 20.

[14] MAGNUS and DE KLEIJN. "Die Abhangigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung," *Pflüger's Archiv*, 1912, Bd. cxlv, S. 455.

[15] SHERRINGTON. "Decerebrate Rigidity and Reflex Co-ordination of Movements," *Journ. of Physiol.*, 1897, vol. xxii, p. 319.

[16] STEWART and HOLMES. "Symptomatology of Cerebellar Tumours," *Brain*, 1904, vol. xxvii, p. 543.

[17] SUTHERLAND and PATERSON. "A Type of Cerebral Maldevelopment (Forebrain Aplasia)," *Quart. Journ. Med.*, 1915.

[18] THIELE. *Proc. Roy. Soc*, 1905, vol. lxxvi, p. 360.

[19] TURNER, VIOLET. "A Case of Prolonged Hyperpyrexia in a Child with a Mid-brain Tumour," *Brit. Journ. of Child. Dis.*, 1916, vol. xiii, p. 261.

[20] WEED. "Observations on Decerebrate Rigidity," *Journ. of Physiol.*, 1914, vol. xlviii, p 205.

RELATION OF THE FORM OF THE KNEE-JERK AND PATELLAR CLONUS TO MUSCLE TONUS.[1]

BY HENRY VIETS, M.D.

Boston.

I.—INTRODUCTION.

VARIATIONS in the form of the knee-jerk under various degrees of muscle tonus have been observed both clinically and in the physiological laboratory. These observations have extended over many years from the clinical deductions of Erb and Westphal in 1875, through the more recent physiological experiments of Sherrington and others. From these studies the clinician has drawn certain practical concepts such as "exaggerated knee-jerks," "spastic knee-jerks" and "spasticity," terms used to denote the stiffness of the muscle and the increase in the amplitude of the reflex response most commonly seen after lesions of the pyramidal tracts. These expressions are used in a rather loose way, simply denoting gross quantitative changes, for the most part not to be measured with much degree of accuracy.

Attempts, however, have been made to measure and record variations of the knee-jerk in man and to show the relations between these variations and muscle tonus. Weir Mitchell and Lewis [11] in 1886 contributed the first of such observations and came to the conclusion that "the responsive jerk brought about by striking a stretched tendon is the most refined means we possess of deciding as to the tone of a muscle." This, I think, has been borne out by research since that date, although we have developed more "refined means" to aid in the decision. Bowditch and Warren [1] in the Harvard Laboratory two years later measured the knee-jerk in man by a cleverly arranged but complicated apparatus. Lombard [4] also carried out experiments on man. Somewhat later Wertheim Salomonson [12] published a paper on reflex studies in man with kymographic records. It was noted by all experimenters on men that numerous difficulties prevented the correct recording of their observations. Most of these workers found great mechanical difficulty in attaching a steady apparatus to the lower extremity. Voluntary movements of the patient or his voluntary inhibition of the reflex also caused great variations in their result. These latter phenomena could not be successfully controlled. In short, all attempts to study any single phase of the knee-jerk in man, such as its form, and to record the observations graphically have been complicated by so many factors which could not be controlled that results from such studies are only partially satisfactory.

---

[1] From the Physiological Laboratory, Oxford University.

In animal experiments, however, many of the extraneous factors mentioned above can be controlled by the experimenter. Unfortunately, except for the work of Sherrington, there have been few detailed studies in animals on the relation of the form of the knee-jerk to muscle tonus. It was the aim of this research, then, to demonstrate in animals just what form the knee-jerk may assume under various degrees of tonus. We only attempt to isolate the knee-jerk nerve-muscle group in a living preparation and study it under various degrees of muscle tonus.

II.—METHOD.

(1) *Preparation of animal and apparatus.*—For a study of the knee-jerk in an animal the nerve-muscle preparation isolated was the anterior crural-vasticrureus group. This was comparatively easy in the cat. The dissection as described by Sherrington [7] cuts all the muscles and nerves of the hip and thigh except the internal and external vasti and crureus muscles and the anterior crural nerve connecting them with the central nervous system. The proximal end of the peroneal nerve was also left for external stimulation. This dissection was usually bilateral, giving two knee-jerk preparations. In all cases both peroneal nerves were isolated. Thus we had an isolated reflex group, free from the influence of muscles acting on the same joint which might interfere with the recording of movement of that joint.

Cats were used for all experiments. The preliminary dissections described above to isolate the knee-jerk muscles were done under ether anæsthesia. The two peroneal nerves were exposed at the same time and glass-shielded electrodes attached to their proximal ends. A faradic current was used for stimulation of these nerves. The feet of the animals were amputated at the ankles and a pin attached firmly through the tendons and periosteum at the distal end of the tibiæ to provide a point of attachment for our recording apparatus. If a spinal preparation was to be used a preliminary laminectomy was done at this stage, exposing the cord without opening the dura at about the tenth dorsal vertebral level. The skin was used to cover these operative defects so as to keep the limbs and back warm All experiments were also performed on a warm table.

After the above steps, decerebration was carried out by using the Sherrington decerebrator. In practically every case decerebration was successful and good decerebrate rigidity was obtained. The animal was then placed on its back surrounded with hot-water bottles to insure a fairly even temperature. The head was slightly elevated. Animals could be kept in good condition for hours, in one case for over twenty-four hours. The legs were fixed by pivot pins through holes drilled in the condylar aspects of the femurs. A thread from the fixed pin at the ankle passed over a series of pulleys to recording levers, making a mark on a smoked kymographic drum. An upward fling of the leg (extension of the leg by contraction of the vasticrureus muscle), when the patellar tendon was tapped, recorded on the drum as an upward vertical mark. Variations in the tone of the muscle were recorded on the drum by the various levels of the marker. Thus a low level on the drum indicated low tonus (a low-tension muscle) [9], while a high level denoted a high tonus (high-tension muscle). Electrodes of the Sherrington [6] bipolar type were then attached to both peroneal nerves by separate circuits for stimulation by the faradic coil.

If a spinal preparation was desired the previously exposed spinal cord was cut entirely through at the tenth dorsal level, and the animal was then turned on its back and the same system of recording the movements of the vasticrureus as above described was used.

(2) *Variation of tonus in the isolated reflex group.*—Sherrington [8] had found that there was a marked increase in tonus after decerebration in extensor muscles such as the vasticrureus. This tonus of an antigravity muscle group can be decreased or increased by the stimulation of a sensory nerve of the same side or opposite side of the body. Thus, if the peroneal (partly sensory) nerve, which is convenient and accessible, be stimulated in the ipsilateral limb the tonus of the vasticrureus group is at once lowered. Vice versa, if the contralateral peroneal nerve be stimulated the tonus of this antigravity muscle group is increased. The relaxation of the extensor vasticrureus when an ipsilateral nerve is stimulated is part of the reciprocal innervation of the flexion reflex. Thus stimulation of ipsilateral and contralateral peripheral nerves enabled us to vary the tonus of this extensor muscle. By cutting the spinal cord in the mid-thoracic region we had a spinal

preparation with flaccid limbs and "toneless" muscles. Thus we could vary our tonus from complete rigidity, sometimes seen in the freshly decerebrate preparations, to flaccidity seen in the spinal animal, with variations between these two extremes by stimulation of the ipsilateral or contralateral peroneal nerves.

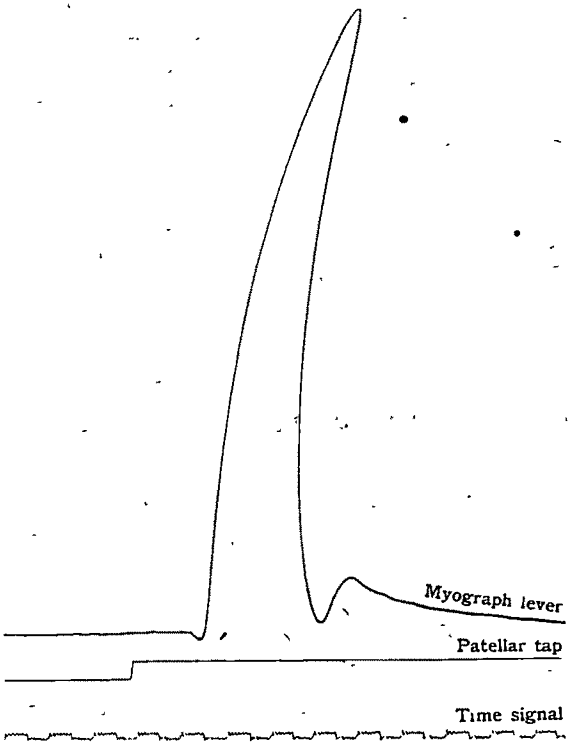


FIG. 1.—Myogram of the form of the isolated knee-jerk of a spinal cat. The upper line records the movement of the vasticrureus muscle when the patellar tendon is tapped. An upstroke indicates contraction of the muscle; a downstroke, relaxation. The approximate time of the blow of the patellar is indicated by the second line. The lower line is the record of a tuning fork vibrating 13·5 times per second.

III.—OBSERVATIONS.

(1) *The knee-jerk in the spinal and decerebrate cat.*—The response to a tap of the patellar tendon in a spinal preparation was first examined. The spinal cord had been cut through at the level of the tenth dorsal segment and the vasticrureus muscle showed very little tone. When the limb was elevated and let fall it would drop back to its original position. There was no "lengthening" or "shortening"

of the muscle on passive movement. After any manipulation the same level as before was recorded on the drum (i.e., the tension of the muscle remained constant). Although the muscle was flaccid to palpation the knee-jerk was fairly active. A unilateral record taken of this preparation is shown in fig. 1. The curve is a simple one of contraction followed by an almost parallel relaxation, the same level being maintained after the jerk as before. There was a slight rebound after the jerk. This rebound was thought not to be due to either mechanical bouncing of the knee-joint or the lever. It was presumed that the sudden elongation of the muscle after its contraction was enough of a stimulus itself to give another knee-jerk. With the cat on its back and the leg only supported by the distal end of the femur, the weight of the leg is, of course, a large factor in its return to its original position after the jerk. The second line of the record indicates the moment a hammer fell from its fixed position just above the patellar tendon. It does not, therefore, represent the exact moment the tendon was hit, for a slight amount of time was taken by the hammer falling a few centimetres. This record cannot be used, therefore, to measure the time of the reflex, as it gives approximately only the exact time of the stimulation.

Fig. 2 is a record of the same decerebrate animal without the spinal cord cut. In this experiment there was a moderate degree of decerebrate rigidity. All the extensor muscles of the upper limb were tonic and the vasticrureus was spastic to palpation. After the limb was extended from a flexed position by hand the muscle continued to stay shortened and showed a higher level on the drum than before it was moved. Vice versa, if the limb was flexed by hand a new level of tone was developed which continued without further change. The limb, therefore, showed the "shortening" and "lengthening" reaction and "plasticity," terms used by Sherrington to describe these phenomena; in short, the vasticrureus muscle could be "set" at various lengths and it would remain fixed at its new length. This plastic element in these spastic muscles was demonstrated in many decerebrate preparations, and we came to recognize it as one of the most constant signs in decerebrate rigidity. In this spastic vasticrureus muscle the knee-jerk showed a form quite different from the non-spastic or spinal muscle. Two elements would be easily recognized: the jerk which appeared first and resembled the jerk of the spinal knee-jerk, and the second factor, the plasticity. The jerk was slightly greater in amplitude than the corresponding jerk in the spinal animal. After the jerk, however, the muscle did not completely relax but remained partly contracted at a shorter length than before it was stimulated. This shortening showed as a higher level on the recording drum. Thus there was a new tonic level after each knee-jerk more tonic than the level before the jerk. The muscle continued at this level or tonic state until another jerk or some other factor changed its tonus. The form of the knee-jerk, therefore, was changed in the decerebrate from the simple jerk seen in the spinal animal to a jerk plus a continued shortening of the muscle, the second element being the result of the "plastic tonus." Fig. 2 is a typical curve taken from many, and when compared with fig. 1 the

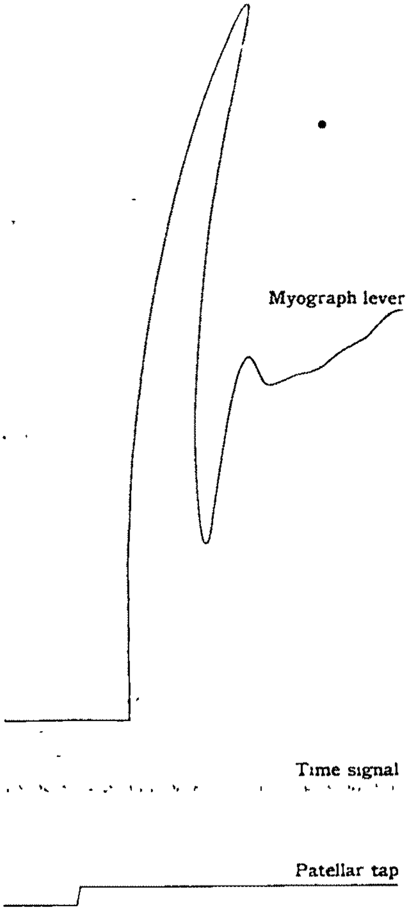


FIG. 2.—Myogram of the form of the isolated knee-jerk of a decerebrate cat. The tracings indicate movements as in fig. 1, except that the time record is placed above that of the patellar tap.

difference in the final phase of the knee-jerk is apparent. In the cat, then, we may say that increased tonus of the vasticrureus muscle (decerebrate) causes a change in the form of the knee-jerk, the jerk being slightly greater in amplitude, and also exhibiting plasticity not found in the muscles with slight tonus (spinal).

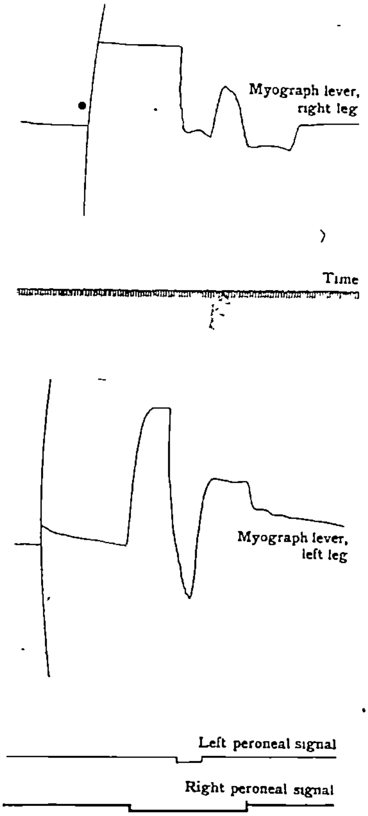


Fig. 3.—Reciprocal innervation in two isolated vasticrurei muscles. (See text for description.) An upstroke of the lever indicates increased tension of the muscles; a downstroke, decreased tension. Time, 13·5 beats per second.

We may vary the tonus of the vasticrureus muscle by stimulating the ipsilateral or contralateral peroneal nerves. Fig. 3 is a record of such an experiment. The movements of the two vasticrureus muscles are recorded as well as the time of the peroneal stimulations. The first nerve stimulated was the right peroneal. The tonus of the right extensor muscle was inhibited by this stimulation and the tension of the muscle relaxed, i.e., the writing point dropped; at the same time the tone of the left vasticrureus muscle was increased, the tension increased and its lever was raised. While the tone of the right extensor muscle was lowered and that of the left raised, we stimulated the left peroneal nerve and the reverse of the above took place; the tonus in the right muscle was increased, and in the left muscle

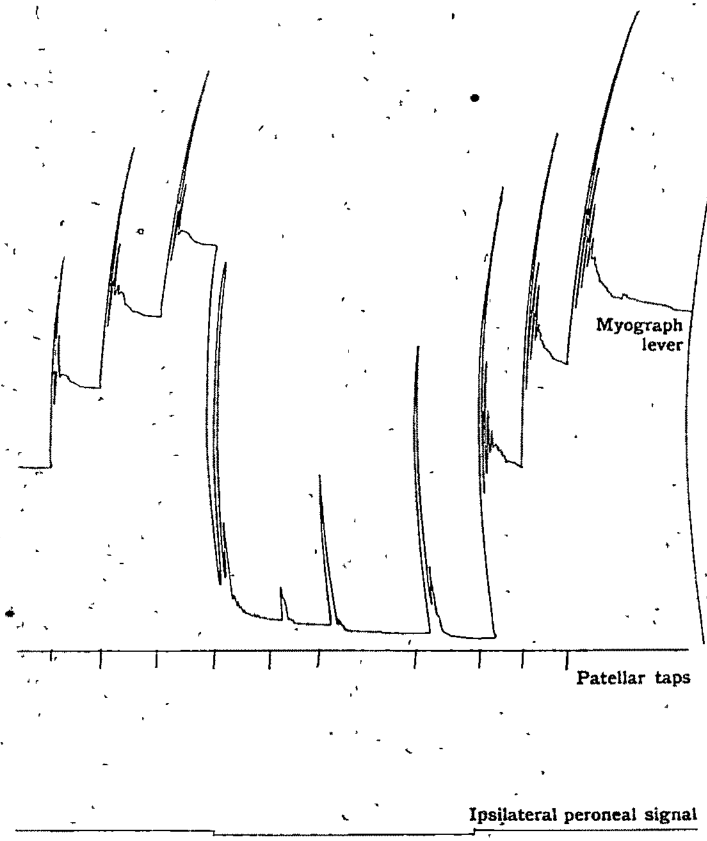


FIG. 4.—Showing the effect of ipsilateral peroneal stimulation on the form of the knee-jerk in a decerebrate cat. (See text for description.)

decreased. As the stimulation of these two peroneal nerves was removed by cutting off the faradic current in each, the original movements described above took place in the reverse order. It is clear, then, that we were able to raise or lower the tonus of these knee-jerk muscles by the use of contralateral or ipsilateral peripheral stimulation.

(2) *The knee-jerk in tonic variations.*—Fig. 4 illustrates an experi-

ment in a decerebrate preparation showing marked spasticity with the usual plastic element. The first three knee-jerks recorded show the increase of tonus after each jerk. This causes a "staircase" effect in the record, a condition characteristic of decerebrate rigidity in the extensor muscles of the cat. The tone of this extensor muscle was then lowered by stimulation of the ipsilateral peroneal nerve, and during this stimulation a number of knee-jerks were elicited corresponding to the spinal type of knee-jerk in each case. Although they varied somewhat in amplitude the relaxation after the jerk was as great as the contraction and the element of plasticity in the muscle has been eliminated. After the current was released the knee-jerk again assumed the form of the decerebrate preparation. Of the ten knee-jerks recorded in this figure the first three and the last three show the typical decerebrate form with plasticity, while the four middle ones show the simple twitch or spinal type. The middle line of the record marks approximately the time of stimulation of the patellar tendon, while the lower line indicates the beginning and ending of the ipsilateral peroneal stimulation. There was a slight tendency to clonus (pseudo-clonus) in all of the decerebrate knee-jerks, not so marked in the spinal ones.

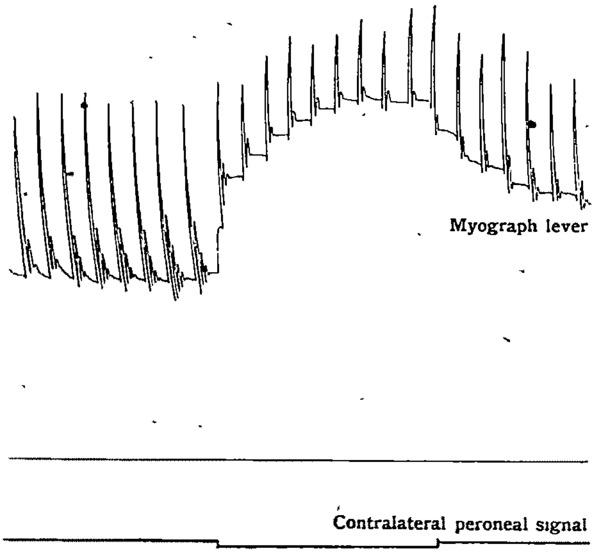


FIG. 5.—Showing the effect of contralateral peroneal stimulation on the form of the knee-jerk in a spinal cat. (See text for description.) Points of patellar taps not indicated.

As fig. 4 represents a spastic or decerebrate knee-jerk taking the form of a flaccid or spinal knee-jerk by inhibition of the tonus, fig. 5 shows the reverse condition, a flaccid knee-jerk of a spinal animal becoming a spastic knee-jerk as in a decerebrate animal. This record

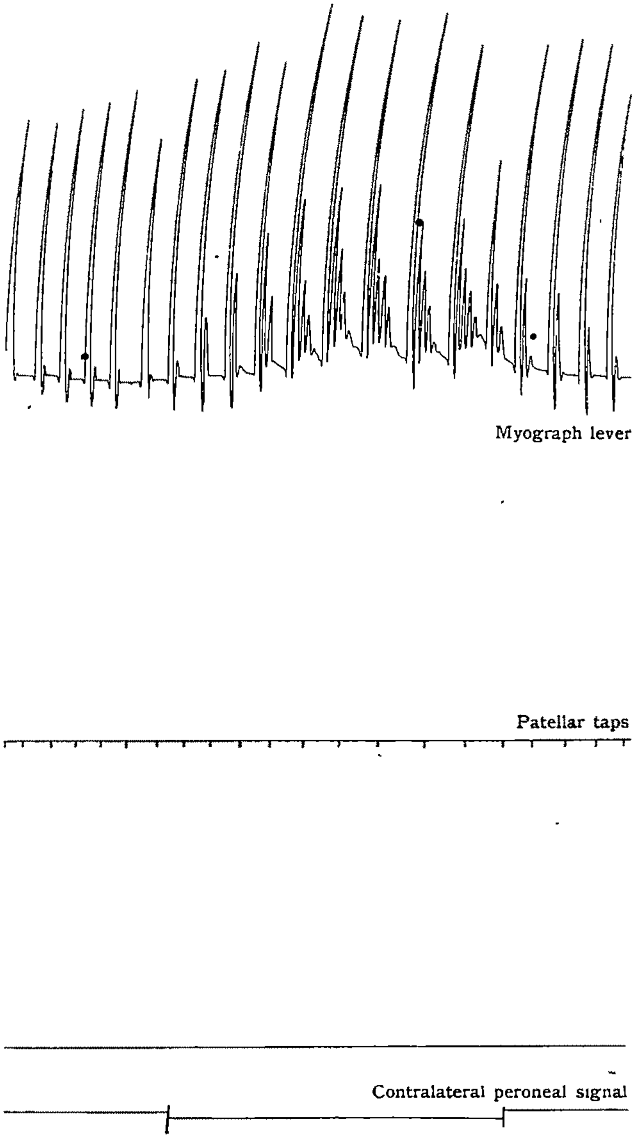


FIG. 6.—Slight increase in tonus with contralateral peroneal stimulation in a spinal cat with development of a pseudoclonus. Middle line indicates points of patellar taps. Lowest line shows point of peroneal stimulation.

is of an experiment on a spinal animal, and it can be seen that the knee-jerks at the beginning of the record are typical of the spinal type. Between the ninth and tenth knee-jerks on the record the contra-

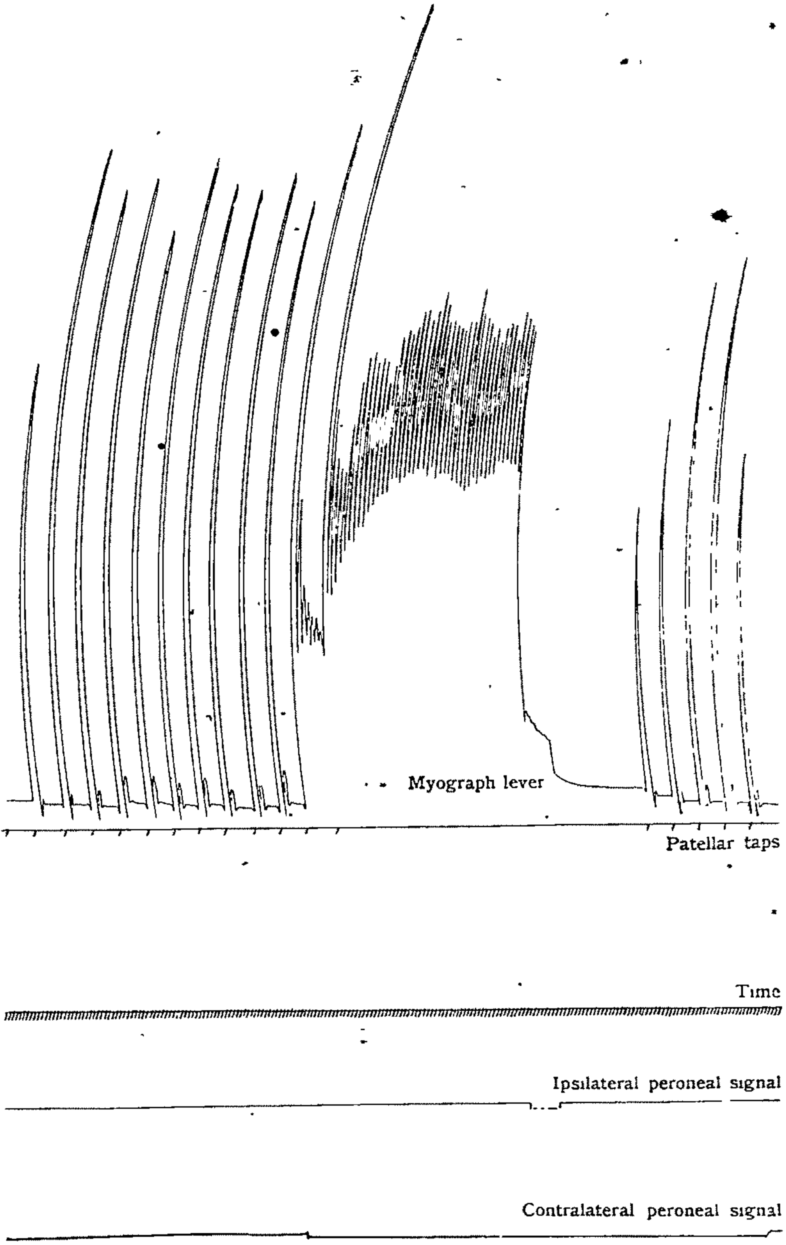


FIG. 7.—Knee-jerks in spinal cat augmented to a clonus by contralateral peroneal stimulation and stopped by ipsilateral inhibition. The first ten knee-jerks are "spinal" in form, At this point contralateral stimulation raised the tonus, and the next knee-jerk was spastic. followed by another with clonus. The tonus was then strongly inhibited by ipsilateral stimulation, the clonus stopped and the knee jerks resumed their spinal form. The upper line is the myograph lever, below this, the signal of the patellar taps, the time 13·5 beats per second, ipsilateral peroneal signal, and below it, contralateral peroneal signal.

lateral, peroneal nerve was stimulated, thus increasing the tonus of the extensor muscle, and during this increase of tone the knee-jerks assumed the spastic form with spasticity. When the stimulation was released, the knee-jerks again resumed their spinal character. The lowest line of the record indicated the beginning and ending of the contralateral peroneal stimulation.

In the last two records, therefore, we have first a preparation with a high degree of tonus and plasticity showing the typical decerebrate knee-jerks reduced by ipsilateral stimulation to a preparation with a low degree of tonicity showing the spinal type of knee-jerk. On the other hand, the second experiment illustrates the exact reverse of this; a spinal animal with a typical spinal knee-jerk has its tonus in the extensor muscle increased by contralateral stimulation, and the knee-jerks show the decerebrate or spastic type while the muscle is thus stimulated.

(3) *Patellar clonus.*—A pseudoclonus was often noted in both the spinal and decerebrate preparations. There was more tendency for it to develop when the tonus was slightly elevated by stimulation of the proximal end of the contralateral peroneal nerve (fig. 6). It will be seen that the knee-jerk showed a slight degree of plasticity during this stimulation, and also that the "rebound" or second jerk became a short series of jerks from two to four in number. If, however, the tonus augmenting stimulus was slightly stronger than shown in fig. 6, a real self-sustaining clonus could be developed, the relaxation after each contraction being enough of a stimulus presumably to set off another jerk (fig. 7). During this experiment, when the tonus was suddenly dropped by an ipsilateral peroneal stimulation, the clonus at once stopped and the knee-jerk resumed its original spinal form.

Such a clonus was often started in a decerebrate animal with a spastic vasticrureus muscle by raising the tonus by the "staircase" method of repeated knee-jerks. Fig. 8 is the record of an experiment showing that one patellar tap was sufficient to cause a pseudoclonus and raise the tonus enough so that the second tap was followed by a continued or actual clonus.

Fig. 9 illustrates an experiment in which the clonus is shown in a little more detail. This tracing, from an experiment on a decerebrate cat with a spastic vasticrureus muscle, illustrates the development of a clonus after a single knee-jerk raised the tonus to a certain level. It also illustrates the regularity of the contraction and relaxation of the muscle during the clonus. The rate of the clonus in

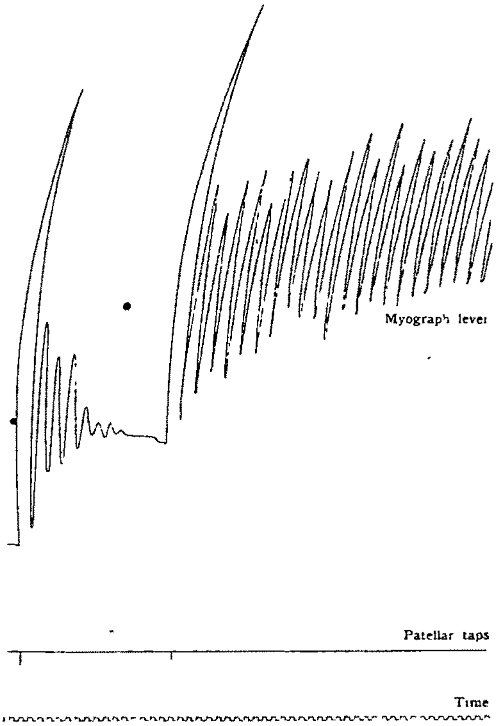


FIG. 8 - Clonus in vasticrureus muscle of decerebrate cat inaugurated by successive knee-jerks, the "staircase" effect The middle line is the signal for patellar taps. The lowest line is the time record, 13·5 beats per second.

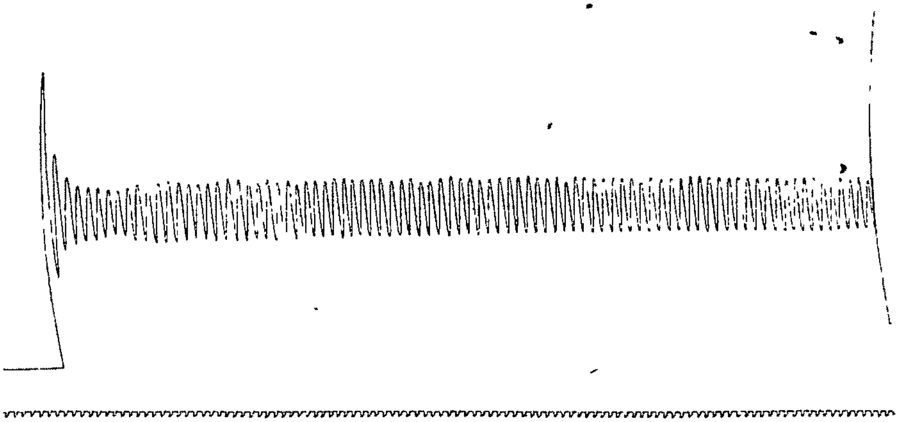


FIG. 9.—Patellar clonus in decerebrate cat, vasticrureus muscle, inaugurated by single knee-jerk. Rate of clonus, 11·5 jerks per second. Time, below, 13 5 beats per second.

this experiment was 11·5 complete contractions per second. Other records of patellar clonus were 11·2, 12·6 and 11·5 complete contractions per second. In still other animals the rate was higher, 17·2, 17·1, 17·3 and 16·4 complete contractions per second. There seemed to be, therefore, two rather definite rates, one about twelve complete beats and another about seventeen complete beats per second. The rate was always nearly constant in the same animal.

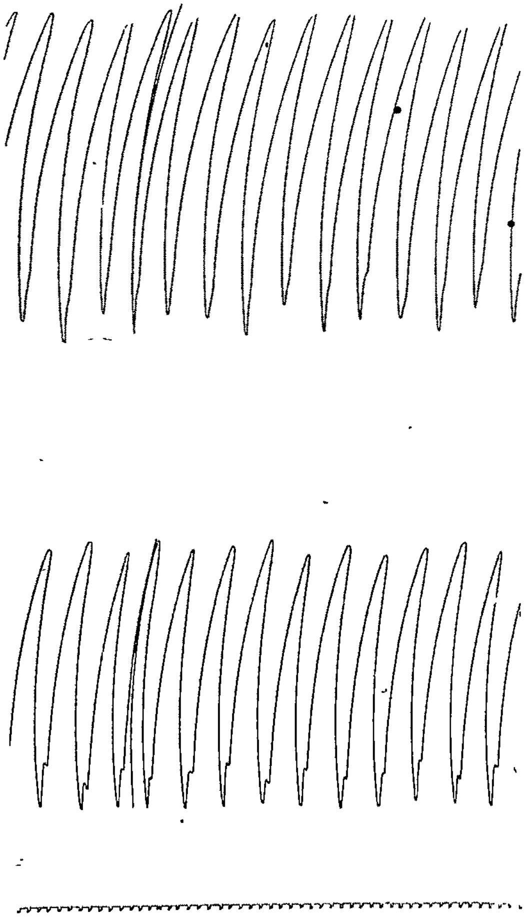


FIG. 10.—Reflex "walking" in a decerebrate cat; two vasticrurei muscle preparations. Alternate contraction of the two muscles. Rate, 3·5 steps per second. (For full description, see text.)

One case of patellar clonus was studied in man, using the same apparatus. The case was one of a hemiplegia of some years' duration with marked spasticity of the left leg, increased knee-jerks, patellar clonus, ankle clonus and a positive Babinski reflex. The rate of the patellar clonus in man was between 8·8 and 9·7 complete beats per second.

By way of comparison with the clonus rate some records were obtained of the "walking reflex," an alternate contraction and relaxation of the vasticrurei, and fig. 10 shows a record of the two extensor muscles during the walking reflex. The rate of this walking at the time this record was taken was 3·5 steps per second, but great variations in the rate were observed. At one time it was recorded that the rate was 1·7 steps per second and at another 2·7. It will be seen, therefore, that the walking reflex is quite different from the patellar clonus. The patellar clonus is very constant in the same animal, although it may be of different rates in different animals. The walking reflex, on the other hand, varies in rate from time to time in the same animal.

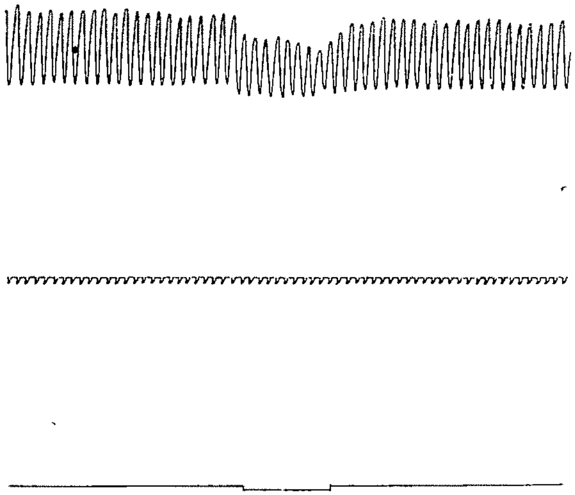


FIG. 11.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus inhibited by ipsilateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 13·5 beats per second.

A double clonus was never successfully recorded in any of the experiments, although it was observed a number of times. The contractions were synchronous, not alternating as in walking. Clonus was not always obtained in decerebrate cats, but appeared at unsuspected times, often when our apparatus was not adjusted for recording owing to mechanical difficulties. We were never able to obtain clonus by any special form of decerebration and it was always observed incidentally while working on the form of the knee-jerk.

Variations in the form of the clonus were observed during changes in the amount of clonus exactly corresponding to variations in the form of the knee-jerk during changes of the tonus. Fig. 11 is a tracing of an experiment in which the tonus was decreased by an ipsilateral peroneal stimulation during the recording of a clonus. The record shows the drop in tone and also that the clonus changed slightly in character. The rate remained the same, but the clonus became irregular with variations in the size of the jerk. When the inhibition of the tonus was released the clonus gradually "climbed" and sought the same level (i.e., tone) as before inhibition. On the other hand, when the tone was increased by contralateral peroneal stimulation the same phenomena were observed (fig. 12). There was a decrease in the amplitude with a slight irregularity. If the tonus was decreased or increased markedly the clonus would at once stop and it was observed that only between certain degrees of tonus (*i.e.*, certain levels on the drum) could clonus be obtained, and any tonus above this level or below this level was sufficient to stop the clonus.

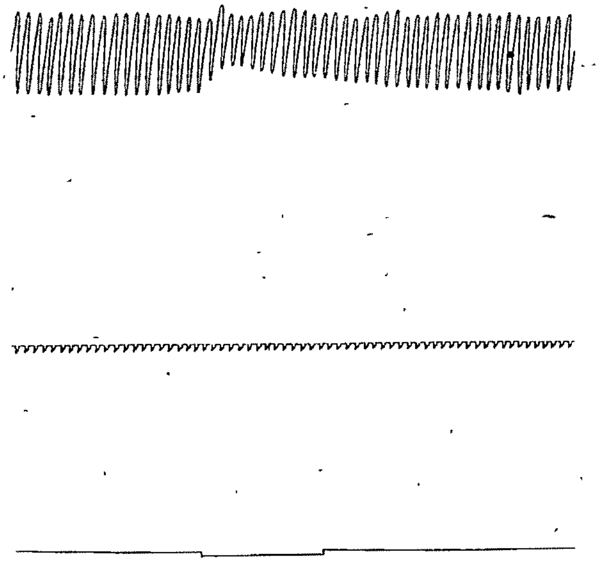


Fig. 12.—Patellar clonus in a decerebrate cat; vasticrureus muscle. Tonus augmented by a weak contralateral peroneal stimulation, indicated by signal, lowest line. Time, middle line, 18·5 beats per second.

IV.—DISCUSSION.

There are two distinct forms of the knee-jerk noted in these experiments. One is the simple jerk with the contraction and relaxation of about the same degree, as seen in the spinal animals. The other is the decerebrate knee-jerk in which in addition to the twitch there is a plastic element which holds the muscle at a new degree of tonic level after the jerk. This second element in the decerebrate knee-jerk was shown by Sherrington [7] in his experiments on the shortening and lengthening reactions. He showed that the spastic muscle in the decerebrate cat was capable of being "set" at various lengths and would remain so for an indefinite period. These two elements in spastic muscles in decerebrate animals have been noted by other investigators. Langelaan [3] proposed the terms "contractile tonus" and "spastic or autonomic tonus," and clearly showed them in his experiments on animals. He concludes "that the contraction following the tap on the tendon is of a twofold nature, viz., a twitch upon which is superposed a tonic contraction."

Plasticity as a part of spasticity has not, so far as I am aware, been adequately demonstrated in man except for Wertheim Salomonson's paper. He obtained, in a case of cerebral diplegia, curves of the knee-jerk exactly corresponding to the spastic curves seen in the cat (fig. 2.) He noted that "the muscle does not entirely relax, but a state of hardening of the muscle substances persists for a longer or shorter time." From the illustrations he uses, the "hardening," I think, is the same as the plastic tonus described by Langelaan and others. The twitch is followed by a continued contraction as in the experiments on animals. I have, however, noted plasticity in some of the severe cases of hemiplegia and in one case of diplegia, but have never recorded this graphically. The so-called "lead pipe" rigidity is, I think, an example of plasticity combined with spasticity in man. Nevertheless it is certainly not the usual finding in a spastic muscle in man, as the most noticeable phenomenon is the great increase in the amplitude of the responses to the patellar tap. Thus the laboratory finding, where plasticity is the usual companion of spasticity, is not always easily demonstrated in the clinic.

In the laboratory, however, we are observing animals whose nervous system is far less integrated than the nervous system of man. The activity of the spinal animal where reflexes may be demonstrated in almost pure form is much greater than the "spinal man." It must be recognized, nevertheless, that many of the reflexes such as the scratch reflex and the extensor thrust, so clearly demonstrated by Sherrington in animals, are present in man, although they may be so weak or changed that they are not easily found. The work of Head and Riddoch [2] on certain reflexes in cases of complete spinal cord lesions without infections shows that many more reflexes may be seen in the isolated human spinal cord than was previously supposed. Riddoch [5] found that the usual knee-jerk in his cases of complete section of the spinal cord was a simple twitch, presumably the same as is shown to be the case in spinal animals (fig. 1). In one of his cases, however, where there was not a complete transection of the cord he found after the twitch of the knee-jerk "the leg fell away more slowly and the relaxation of the tendon was more deliberate." He thought in this case there was some "plastic tonus," as well as "contractile tonus," to use Langelaan's term. Walshe [10] has also successfully searched in men for reflex actions that Sherrington had demonstrated in the laboratory.

It is, perhaps, not remarkable that the plastic element in spasticity has not been observed more often in man. It must be remembered that one seldom has a chance of seeing in man an isolated reflex group such as is artificially produced in animals. Also, the extraneous factors which I mentioned in the early part of this paper are so profound that one cannot observe a pure reflex contraction without the action of other muscles on the same joint or inhibition or augmentation of the reflex by voluntary control. It was observed in these experiments that it was not possible to obtain a knee-jerk in an absolutely toneless muscle. In the spinal animal the vasticrureus muscle was not entirely toneless, for we were able to inhibit some tonus by ipsilateral peroneal stimulation. In other words, we were able to decrease its tonus and finally a point was reached where no reflex was obtained. This, I think, demonstrates that tonus is not entirely dependent on control from centres in the mid-brain, for when the spinal cord is entirely cut across we have severed all connection with such centres. Such a spinal animal has very little tone and we speak of the muscles as flaccid, but nevertheless there is some tonus left. This point, too, has been demonstrated in man by reflexes obtained after complete transverse lesions of the spinal cord. Such reflexes I believe are not possible without some tonus.

It was noticed that the spastic vasticrureus muscle when "set," well lengthened, and then stimulated by blows on the patellar tendon, tended to shorten at each jerk until a certain level was reached on the recording drum, and then tended to remain at that level. In other words, the muscle found a length between its high and low tonus length, at which point there was a marked tendency to remain. This level was also shown in the experiments where the tonus was decreased or increased by ipsilateral or contralateral peroneal stimulation; after such stimulation the muscle took what I have called a "neutral tension." Two factors seem to control this tension, one the reflex tonus tending to increase the tension of the muscle and the other the weight of the limb tending to pull it down and thus lengthen the muscle. When the reflex tonus was very great the muscle was shortened as far as the knee-jerk would allow, the limb being completely extended and the weight of the limb being completely overcome. The point of interest is that at a certain level of tonus, clonus was most apt to appear. It is at the neutral tension of the muscle somewhere between its high and low tonic level that clonus is most apt to appear. In other words a muscle at this tonic tension is in its best position to maintain a continued series of contractions. Clonus was obtained mainly in the spastic muscles at their neutral tension. If a spastic vasticrureus was set at a low level of tonus and the patellar tendon tapped, the plasticity after the jerk brought the muscle to the most favourable level before clonus would appear. If the tonus during a clonus was increased or decreased by contralateral or ipsilateral peroneal stimulation and passed beyond the narrow limits of its neutral tension the clonus either became irregular or disappeared. Langelaan has noted that "a clonus is composed of a series of twitches superposed upon a tonic shortening." This, I think, is correct in a measure, but the tonic shortening (or increased tension) must be a definite one, for any increase or decrease in the tension of the muscle beyond the narrow limit of its neutral tension is enough to cause the clonus to stop. Wertheim Salomonson found clonic after-contractions (pseudoclonus) in many of his cases and in others stated that "the tonus is not quite high enough to permit of a clonus." He showed no records of a true clonus.

It would seem then that a true clonus is a series of continuous reflex twitches dependent upon a more or less fixed tone of the muscle. Clonus is possible only over a slight range of tonus, for there is a tonic tension of a muscle, a "neutral tension," that is, the tone "of choice," for a series of continued self-sustaining twitches. If the plastic element holds the muscle at this length, then the contractile force will produce the jerk, and a clonus will result.

The clonus, as we have noted, is a regular series of twitches. If the tone is raised or lowered, the clonus becomes irregular in both amplitude and rate. Thus we have in our plastic tonus the mechanism by which such a continued muscular effort as the clonus is controlled, so that it continues regularly and smoothly. The tonic level is so regulated by plasticity that the muscle is set at a correct height, so that the falling limb is just enough stimulus to provoke the next twitch. This seems to me one of the functions of plasticity, and its value in animal economy cannot be doubted.

CONCLUSIONS.

Experiments on cats, both spinal and decerebrate, with the anterior crural-vasticrureus nerve-muscle preparation isolated in situ, and the movement of the knee-joint recorded graphically, gave the following results:—

(1) In the records from the spinal animal the knee-jerk is a single twitch, the contraction and relaxation of the extensor muscle group being almost parallel. After the jerk there may be another jerk ("rebound") or a short series of jerks ("pseudoclonus"), but the length of the muscle after each jerk is the same as before the experiment.

(2) In the records of the decerebrate animal the knee-jerk is a single twitch followed by a continued contraction, the muscle assuming a new and shorter length after the jerk. Thus, there are two elements in the phenomenon, the twitch or jerk and the continued contraction. The first may be termed the "contractile element," and the second the "plastic element," after Langelaan.

(3) The plasticity is an indicator of muscle tonus. When the tonus is varied by artificial means, the amount of plasticity varies also. When the tonus is lowered by an ipsilateral peripheral nerve stimulation, the plastic element may drop out of the knee-jerk record, and the jerk takes the form seen in the spinal animal.

(4) Some tonus can be demonstrated in the flaccid vasticrureus muscle of the spinal animal, for it may be reduced by ipsilateral peripheral nerve stimulation, showing that the distal portion of the completely severed spinal cord in a cat is capable of maintaining some tonus in the muscles of the lower limbs.

(5) Many of the knee-jerks in both spinal and decerebrate animals may show pseudoclonus; but it is most often found in decerebrate preparations, showing little tonus.

(6) A true clonus may be obtained in various ways. If the tonus of a spinal animal is raised by contralateral peripheral nerve stimula-

tion, a clonus may result. Decerebrate animals with moderate tonus most often exhibit clonus. The degree of tonus of the muscle at the time of clonus is termed its "neutral tension." This tension is a neutral point between the reflex tonus, tending to increase the tension of the muscle and weight of the limb tending to lengthen it. This tension, and this tension only, is the most favourable point for the maintenance of a clonus.

(7) A true clonus is characterized by a continued series of self-sustained reflex twitches, regular in rate and amplitude, markedly altered or stopped by increasing or decreasing the tonus.

(8) The rate of a clonus in the vasticrureus muscle group of a cat was found to vary, most of the records falling into two groups—a rate about 12 per second and another about 17 per second. Patellar clonus in man was found to be 8·8 to 9·7 per second.

(9) The rate of "walking" varied from 1·75 to 3·5 steps per second.

BIBLIOGRAPHY.

[1] Bowditch and Warren. *Journ. Physiol.*, 1890, vol. xi, p 25.
[2] Head and Riddoch. *Brain*, 1917, vol. xl, p. 188.
[3] Langelaan. *Brain*, 1915, vol. xxxviii, p. 235.
[4] Lombard. *Journ. Physiol.*, 1889, vol. x, p. 122.
[5] Riddoch. *Brain*, 1917, vol. xl, p. 264.
[6] Sherrington. *Journ. Physiol.*, 1909, vol. xxxviii, p. 375.
[7] *Idem.* *Quart. Journ. Exp. Physiol.*, 1909, vol. ii, p. 109.
[8] *Idem.* "Integrative Action of the Nervous System," 1906.
[9] *Idem.* *Brain*, 1915, vol. xxxviii, p. 210.
[10] Walshe. *Brain*, 1914-15, vol. xxxvii, p. 269.
[11] Weir, Mitchell and Lewis. *Trans. Assoc. Amer. Phys.*, 1886, vol. i, p. 2.
[12] Wertheim Salomonson. *Proc. Roy. Acad.*, Amsterdam, 1915, vol. xvii, p. 885.

ALTERATIONS OF THE GOLGI APPARATUS IN NERVE CELLS.[1]

BY WILDER G. PENFIELD, M.A., M.D.

RETISPERSION.

AN intracellular reticulum was first described by Golgi [10] in 1898, in the cells of the central nervous system. This observation has been greatly extended, first by his pupils in Pavia, and subsequently by a large field of workers, until at the present time the structure has been described in the cytoplasm of practically all types of cell throughout the animal kingdom [24, 6, 7, 21]. The structure has received a variety of names, such as the internal reticular apparatus of Golgi [10], the "Binnennetz" of Kopsch [15], the canalicular apparatus (Bensley [1]), &c.

This study is concerned only with the apparatus as seen in nerve cells, where it appears in its most highly differentiated form and presents normally great variation in appearance. There have been numerous theories as to its function in the cell. But before a study of this problem can be logically undertaken, an inquiry must be made into the alterations of this structure in varying nerve cell conditions. Such an inquiry is the purpose of this paper.

The Golgi apparatus presents normally, in the great majority of cases, a complete attenuated reticulum with many varicosities or lacunæ (figs. 1 and 2). These varicosities may be homogeneous and black, as though full of some substance in which the silver is deposited, or they may frequently appear empty of such substance, and only the surface or boundary of the space take on the silver. The structure is confined to the cytoplasm, never encroaching on the nucleus or the periphery of the cell. Threadlike prolongations may pass outward into the dendrites, but I have never seen them in the axone process, nor have I found any such description in the literature.

Variations from the complete reticulum are most frequently seen in the spinal ganglion cells. The whole structure appears rarely in one half of the cytoplasm only. It may also, occasionally, be fragmented to a greater or less degree. It may be hypertrophied or meagre, but under normal conditions the general pattern is surprisingly constant for each type of nerve cell.

---

[1] This research forms part of the work done for the degree of B.Sc., in the Physiological Laboratory, Oxford University. Expenses were defrayed partially by the Christopher Welsh Scholarship Fund.

A Zeiss objective, homog. immers. 1·5 mm., and a 6 compensating eye-piece (magnification 1,000) were used for all drawings, which were made with the aid of an Abbé camera lucida, Zeiss. All sections were prepared by the Cajal uranium-formol-silver method without counterstain. All material was obtained from cats.

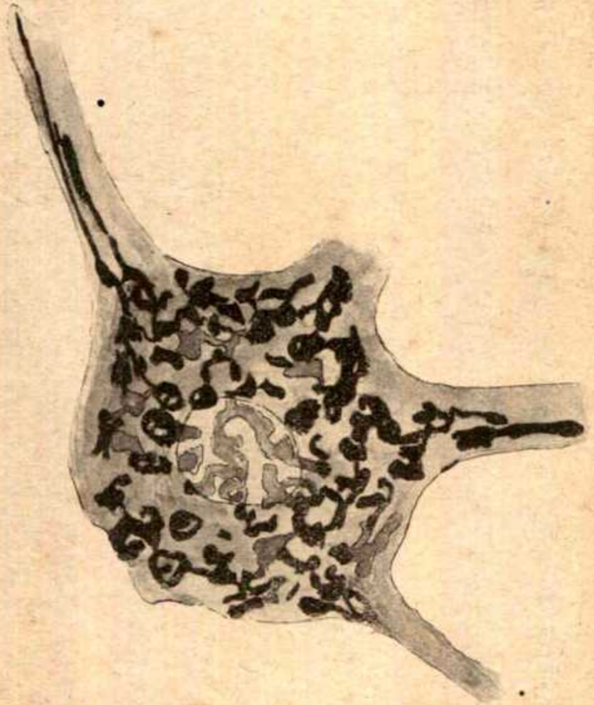


FIG. 1.—Normal cell in anterior horn of spinal cord. That part of the Golgi apparatus which is grey instead of black was drawn with the focus at a deeper level, below the nucleus.

THE LITERATURE OF ALTERATIONS IN THE GOLGI APPARATUS.

We have to thank the brilliant researches of S. Ramón y Cajal and his pupils for the larger part of our knowledge of this subject.

Autolysis.—Autolytic changes in the Golgi reticulum are rapid, beginning in man, according to Cajal, two hours after death. At twenty-four hours the reticulum has become granular, and in some cells only fine fragments will be found to remain.

In the cells of transplanted ganglia, the apparatus shows similar changes [23, 24].

Trauma.—In 1914, when studying the changes following traumatic inflammation, Cajal [23] concluded that the reticulum is more resistant to pathological agents than are the neurofibrils. But he found in the nerve cells of the cord and the pyramidal cells of the cortex that direct trauma causes fragmentation of the reticulum and its displace-

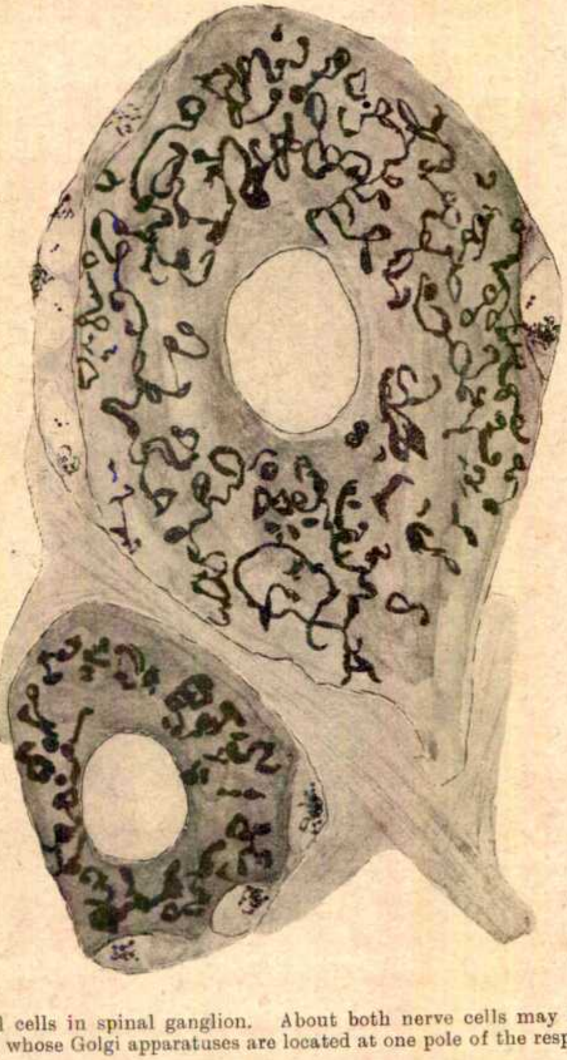


FIG. 2.—Normal cells in spinal ganglion. About both nerve cells may be seen the subcapsular cells, whose Golgi apparatuses are located at one pole of the respective nuclei.

ment to the cell periphery and into the bases of the dendrites. The cell undergoes a process similar to chromatolysis, and as the nucleus approaches the periphery fragments of the Golgi apparatus frequently gather near it. The reticulum may disappear altogether. Therefore, in chromatolysis subsequent to direct injury, the Golgi apparatus and Nissl granulations behave somewhat similarly, both moving out to the periphery of the swollen cell and sometimes disappearing. In later stages, six to twelve days after the wound, he found on its border nerve cells with frankly hypertrophied Golgi apparatus.

Faradism.—Legendre [16] stimulated the posterior root of a lumbar spinal ganglion. He found that, as a result, the Golgi net in each cell was displaced to the periphery and became somewhat deformed. It is not stated what precautions were used to prevent spread of the electrical current along the root to the ganglion direct. Holmgren [12, 13] also tried the effect of electrical stimulation on nerve cells and found that, accompanying chromatolysis of the Nissl bodies, there resulted (*a*) "Kanalizirung," an emptying of the canals, and (*b*) diminution in size of the trophospongium, a system of canals having extracellular connections, which he maintains [14] is identical with the Golgi apparatus.[1]

Lesion of Axone.—Marcora [17, 18] found that, subsequent to section or tearing out of the hypoglossal nerve in a rabbit, there developed a fragmentation and displacement of the intracellular apparatus toward the periphery, which was well marked on the fourth day and reached its height between the fifteenth and eighteenth days. He also noted hypertrophy of the Golgi apparatus after the immediate effect of section had passed.

Cajal [24] described lobulation and fragmentation of the motor cells of the spinal cord after section of the corresponding nerves. He stated that this change accompanied chromatolysis, and, as the nucleus approached the cell periphery, the remains of the broken reticulum often gathered about it, or in the bases of the dendrites. He found no change in the Golgi apparatus of the cells of Clarke's column after section of their axones and, similarly, after section of the sciatic, he reported the spinal ganglia of that side unchanged. He concluded that section of the peripheral extension of a sensory neurone is not a sufficient cause to produce noticeable changes in the Golgi apparatus of that cell.[1]

---

[1] The question of the identity of these two structures is discussed in a paper to appear shortly [22]. For an excellent summary of the literature on this subject, see Duesberg [6, 7].

It is of interest that Cajal [25] showed changes of the internal reticulum in the cells of the sheath of Schwann on the peripheral stump of the nerve after section. The reticulum became increased in quantity, occupied almost the whole width of the sheath, and extended along the fibre in either direction from the nucleus in parallel lines.

NISSL GRANULATIONS—CHROMATOLYSIS.

Inasmuch as many of the changes reported below in the Golgi apparatus are associated with somewhat similar changes in the Nissl granulations (chromaphil substance), it may be of interest to review briefly our knowledge of the latter and the inter-relation of the two substances.

The most delicate morphological index of the functional activity of a nerve cell is generally considered to be the appearance of the chromaphil substance in the cytoplasm, the so-called Nissl granulations.

Alterations in the appearance of this substance caused by electrical stimulation (Nissl, Vas, Lambert, Mann, Lugaro, Pugnat, and others) consist in a progressive diminution associated, in some cases, with displacement of the granules toward the periphery of the cell. Faradic stimulation creates abnormal conditions, however, which are probably not reproduced in the physiological activity of the nerve cell.

The results obtained by a long series of investigators (Van Gehuchten [30], p. 300) indicate that, following normal cell activity, there is a diminution in the amount of Nissl substance, which grows more marked with the onset of fatigue.

After section of the nerve, there may result, as first pointed out by Nissl [20], a change in the cells, the most prominent features of which are: disappearance of the Nissl substance, a considerable increase in the size of the cell, and displacement of the nucleus toward the cell periphery. This phenomenon was called by Marinesco chromatolysis, inasmuch as the alteration in the Nissl substance seemed to be its most striking characteristic. The first signs of change may appear as early as forty hours after the section. (Van Gehuchten [30], p. 305.) The Nissl granules begin to disappear at the centre of the cell, and are visible last about the periphery and sometimes close to the nucleus. Following this phase of "dissolution," which reaches its height after fifteen to twenty days, there ensues a phase of "reformation" which progresses rather more slowly until the cell is again normal in appearance. A certain proportion of cells never recover, but go on to atrophy. The duration and degree of chromatolysis depend on the type of injury to the axone. If the nerve be torn out, the reaction is more severe and certain than that which follows section, ligature, or pressure of the nerve.

---

[1] Page 69 of reprint from Reference 24. "Debemos, pues, concluir, que la sección de la rama periférica de la neurona sensitiva no es causa suficiente para provocar perturbaciones marcadas en el aparato de Golgi de esta célula."

In this paper we are most interested in the cells of the spinal cord and ganglia. The motor cells of the anterior horns of the cord appear to offer considerable resistance to section of the axone. At times there is no resultant chromatolysis (Van Gehuchten [29]). After a lesion of the peripheral neurone-outgrowth of a spinal ganglion cell, chromatolysis is rapid, but section of the central fibres gives rise to no evident reaction. Section of the spinal cord produces in the cells of Clarke's column, located below, an energetic and typical response which is eventually followed by complete atrophy of at least the large cells in the column. (Sherrington and Laslett [28].)

THE GOLGI APPARATUS AND NISSL GRANULATIONS.

In 1910 Legendre [16] proposed that these two structures were identical. He called attention to the fact that, in general, their distribution was the same, both being situated in the cytoplasm, separated from the cell periphery by a clear space and avoiding the axone hillock. When the Nissl granulations were small in size, the Golgi net was composed of small granules and fine filaments. After electrical stimulation of the nerve, he noted similar alterations of the two structures. In transplanted ganglia, he observed the disappearance of the net and granulations at about the same rate.

This close association of the two structures is of interest, but that Legendre was mistaken as to their identity has been amply proven by the observations of other workers. Collin and Lucien [4] impregnated the Golgi apparatus and stained the Nissl granulations in the same sections. They found a certain number of cells where the one was perinuclear and the other peripheral. Marcora [17] stained the two structures simultaneously and demonstrated the Nissl bodies lodged in the mesh of the Golgi net; and in embryonic tissue [19] he showed that the two were separated by a clear unstained space. Cowdry [5] demonstrated the independence of Nissl bodies, "canalicular apparatus," and also mitochondria and neurofibrils in the same cell, by the Bensley method.

Cajal [24] agrees to the similarity of distribution of the two protoplasmic structures with regard to the peripheral freedom of the cell from both and the resemblance of a fragmented reticulum to the Nissl granules. But he calls attention to the fact that, whereas the Nissl bodies are found in the nerve cells only, the reticulum of Golgi is found generally in cells of all descriptions. He reports that his preparations, uranium nitrate-silver impregnation followed by staining of the chromophil bodies with aniline dyes, show constantly a clear space surrounding the Golgi reticulum and separating it from the Nissl substance. He states that this clear space is certainly occupied by neurofibrils.

TECHNIQUE.

In the following experiments the Golgi apparatus was demonstrated by Cajal's uranium-formol-silver method [24],[1] with several minor modifications, as follows:—

The tissue was removed from the body immediately after death. Blocks of the cord must be very thin, not over 1 to 2 mm. After an hour in the fixative, blocks may be removed and cut down more easily to the desired thickness.

(1) Fixation for twelve hours. Shake from time to time. Uranium nitrate, 1 grm.; commercial formol (40 per cent.), 20 c.c.; distilled water, 80 c.c. The formol should be neutral. It is sufficient to keep chalk in the bottom of the container. For the cord and spinal ganglia 20 c.c. of formol gives more consistent results than 15 c.c. as suggested by Cajal. It is best to make up solutions of formol and of uranium nitrate separately, of twice the required strength, and mix in equal parts, just before use.

(2) Impregnation for forty-eight hours. After washing in distilled water for a few seconds only, immerse in 1·5 per cent. silver nitrate. Shake from time to time.

(3) Reduction for twelve hours. (First wash in distilled water.) Hydroquinone, 2 grm.; formol (commercial), 20 c.c.; sodium sulphite, 0·5 to 1·5 grm.; distilled water, 80 c.c. Sufficient sodium sulphite should be added to turn the solution quickly to a coffee colour.

(4) Dehydrate very rapidly in alcohol. Pass through xylol to wax.

---

[1] An excellent summary of this method has been published in English by Carleton [2].

From 90 per cent. alcohol until the tissue is embedded should not occupy more than five hours. If this stage is not carried out rapidly it may be found that all silver will have disappeared from a peripheral zone of cells in the spinal ganglion sections.

The fixation is apparently not sufficient to prevent the subsequent solution of some or all of the constituents of the Golgi apparatus in strong alcohol (Gatenby [9]), if exposed to it for too long a time. Toning of the sections with gold chloride adds nothing.

In two experiments I obtained the best results and perfect fixation of the cord by subarachnoid injection of the fixative before death, as follows: The animal is anæsthetized and a laminectomy performed in the lower lumbar region, exposing the membranes. A medium-sized lumbar puncture needle is then passed into the subarachnoid space a short way upward beside the cord and the fixative allowed to flow in under a gravity pressure of 75 cm. The heart stops after about a minute of the injection, which is continued for twenty hours. At the end of this time the cord is removed, blocks cut and dropped directly into the silver nitrate bath. Likewise, sections from tissue which has been fixed in this way and left a few days in 95 per cent. alcohol give the most perfect result when stained by Nissl's method. Subarachnoid injection does not fix the spinal ganglia properly, if at all.

This method, in general, if adapted to the type of cells one wishes to study, gives very complete and fairly consistent impregnations of the Golgi apparatus. In my experience, strength of fixative and period of fixation are the most important elements in the adjustment of the method to any particular tissue. In the sections prepared for this study there was also impregnation, at times, of the mitochondria in the nerve cells, and the Golgi apparatus in both the neuroglia cells of the cord and the intracapsular cells (Holmgren's "trophocytes") of the spinal ganglia. In the ganglion cells an intra-nucleolar body (*see* Carleton, [3]) was frequently impregnated.

When counterstain was desired, Unna's polychrome methylene blue was employed, demonstrating Nissl granulations, nuclear structures, &c.

For the purpose of this research, cats were used, under two to three months of age as a rule. In the few experiments where older cats were used, the impregnation of the Golgi apparatus (contrary to the experience of Cajal) was equally satisfactory. The frequent presence of well-stained pigment granules, however, in the material from the older animals, added another cytoplasmic constituent.

RESULTS OF EXPERIMENTS.

To reconsider briefly some of the foregoing paragraphs: As a result of section of the axone, changes in the Golgi apparatus have been described in the motor cells of the cord and bulb, but none in the cells of the spinal ganglia or Clarke's column. The nature of the reported change is progressive fragmentation and lobulation of the reticulum with some peripheral displacement. Chromatolysis appears in the cells of the anterior horn and Clarke's column after axone section, and in spinal ganglion cells after section of the peripheral extension of the axone only. Cutting of the central extension fails to cause the reaction. My observations give no cause to modify these conclusions, at least in so far as the phenomenon of chromatolysis is concerned.

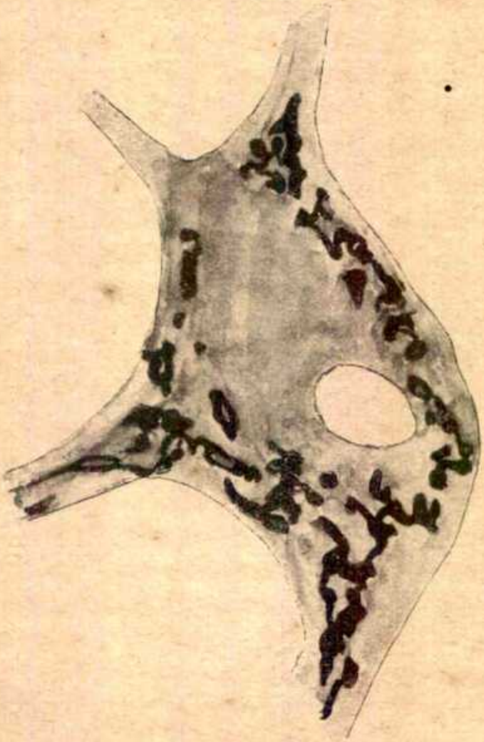


FIG. 3.—Retispersion in anterior horn cell of left side of spinal cord. Level of 7th lumbar segment. Seven days after section of left sciatic nerve.

Positive results.—The work reported here indicates that the Golgi apparatus in all types of neurones studied, afferent as well as efferent, responds to an axone lesion in a consistently specific manner.

The *motor cells* of the cord were studied after simple section of the sciatic nerve. There was no possibility of trauma as the section was made at the level of the great trochanter, a distance of about 5 cm. below the seventh lumbar ganglion when measured along the nerve. At the end of seven days, after the cutting of the nerve on one side, the reticulum had begun to leave a clear zone about the nucleus of the motor cells on that side. This could be made out in 22 per cent. of the cells counted. No such condition was found in the examination of an equal number of cord neurones on the opposite side, where the nerve was intact. Aside from this peripheral displacement, there was very little change in the appearance of the apparatus (fig. 3). After sixteen days the change had become more general, the clear perinuclear zone was wider and now appeared in 50 per cent. of the homolateral motor cells, the heterolateral side being 100 per cent. normal. There was now a moderate amount of fragmentation in some cells, while in others the reticular structure remained unbroken. The neighbourhood of the axone base becomes free of all Gogli apparatus early in the process.

It seems desirable to call this process by a distinctive name, *retispersion* (from *rete*, a net, and *spargo*, to scatter), especially since it may appear independently of chromatolysis as will be shown below. After this stage of dispersion of the Golgi apparatus to the cell periphery, there may succeed a stage of dissolution, when the reticulum becomes progressively fragmented. Thus *retispersion* may be followed by *retisolution*. It seems evident, however, that the amount of retisolution varies with the different types of neurone.

The cells of *Clarke's column* respond in a most energetic fashion. In Experiment 14, the spinal cord of the cat had been completely severed four days previously. The section was at the level of the eleventh dorsal spine. In the lower lumbar region, the cells which correspond to Clarke's column higher in the cord showed a striking change. The Golgi reticulum was close against the periphery of the cell except where some remained about the nucleus, which also had become eccentric. In spite of its extreme dislocation, however, the network of the apparatus was complete for the most part (fig. 5). Retisolution had begun only in a few cells (fig. 6). Nissl granulations were absent.

The *spinal ganglion* cells present normally such a rich variety of types of Golgi apparatus that considerable care must be taken to control all conclusions. Four days after section of the left sciatic, retispersion had begun in the spinal ganglion cells of the same side. The Golgi apparatus had moved outward toward the periphery, leaving a clear space about the nucleus in 48 per cent. of these cells, whereas in the same ganglion on the opposite side the apparatus was more or less peripheral in only 16 per cent., which variation is about the average for

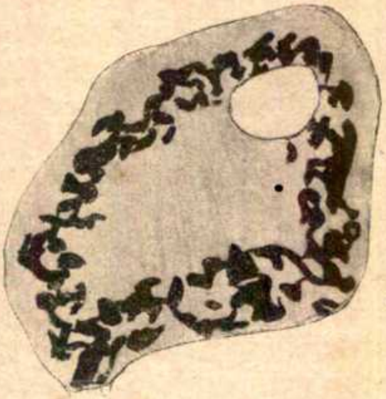


FIG. 5.—Retispersion in neurone of Clarke's column in lumbar spinal cord. Four days after section of spinal cord at level of 11th thoracic spine.

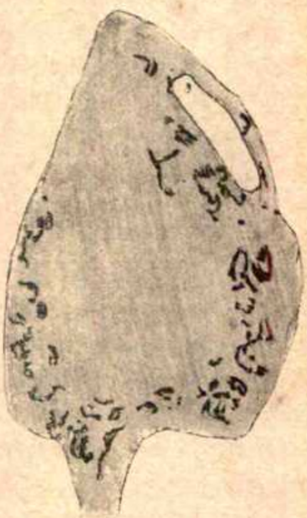


FIG. 6.—Same as fig. 5, showing considerable retisolution as well.

normal ganglion cells. The difference,[1] of course, is more especially one of degree of displacement, which cannot be estimated in figures. Nissl stains showed no evidence of chromatolysis as yet.

---

[1] If sections are cut thin, at about 6 μ, retispersion is less apt to be overlooked than when the sections are thick.

Seven days after the sciatic was cut, the peripheral dislocation of the reticulum reached its height, affecting 80 per cent. of the homolateral cells as compared with a count which was again 16 per cent. for the opposite side (fig. 4). Now, however, fragmentation of the apparatus had begun somewhat and also chromatolysis was present in many of the cells. On the sixteenth day chromatolysis was at its height, but retispersion seemed to have passed its apex. Although in certain cells

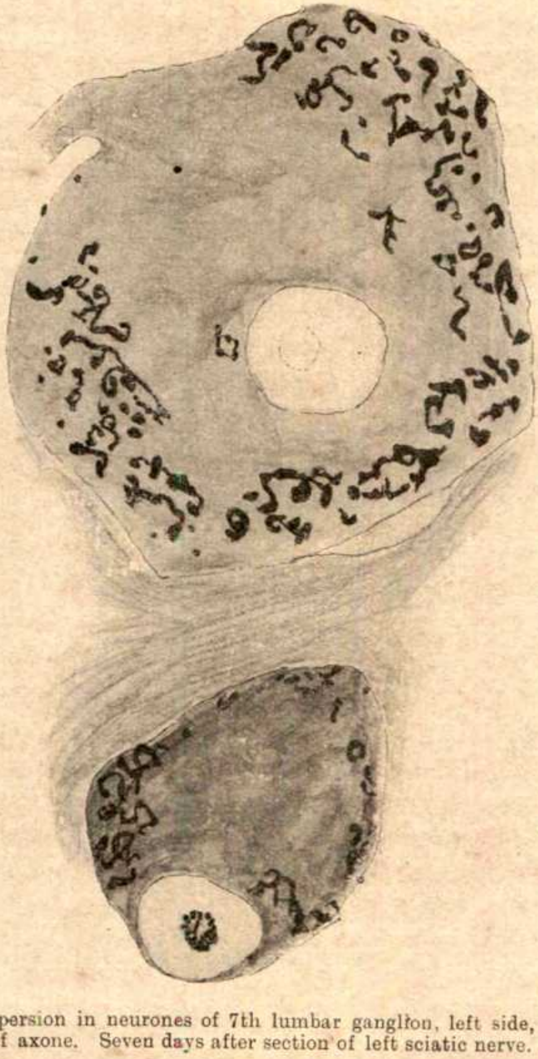


FIG. 4.—Retispersion in neurones of 7th lumbar ganglion, left side, showing the clear space about base of axone. Seven days after section of left sciatic nerve. Same experiment as that of fig. 3.

retisolution was more extreme than in any seen previously, there was a larger proportion of cells with a complete Golgi apparatus in its normal position than on the seventh day. In the larger cell (fig. 4) the absence of reticulum from the neighbourhood of the axone is striking. In general, that part of the cell which becomes free from all Golgi apparatus has the shape of a pear whose stem corresponds to the axone (see also fig. 3).

Interest naturally turns next to an inquiry as to whether cutting of the central extension of the axone of the spinal ganglion cell is followed by retispersion. According to Van Gehuchten, as has already been stated, this cutting of the central fibres is, curiously enough, not followed by chromatolysis ([30], p. 316). The posterior nerve roots proximal to the sacral and lower lumbar spinal ganglia were cut. Seventeen days later, retispersion was present, although not extreme. Double staining by the Cajal and Nissl methods showed the Nissl bodies for the most part normal in amount and location, although in a few cells they could be stained only at the cell periphery in the mesh of the Golgi apparatus.

The attempt was next made to produce retispersion in those sensory cells some of whose fibres pass upward through the cord without interruption. The spinal cord was cut and the ganglia lying below examined; but no conclusive evidence of a resultant change could be made out two, four, six, or twelve days after section or semisection of the cord. It must be borne in mind, however, that only a part of the central neurone-outgrowth could have been thus severed. Other divisions of the neurone end below the site of section.

Negative results.—Four and twelve days after section of the cord above, no change was found in the Golgi apparatus of the anterior horn cells. Seven days after an aseptic decerebration performed on a cat by H. C. Bazett (to whose kindness I am indebted for the material), no changes could be found in the cord motor cells. One must conclude that the alterations in the activity of the reflex arc following cord section or decerebration, are not reflected by any detectable change of the Golgi apparatus in the motor cells.

Local tetanus was produced in one hind limb of cats by opening the sheath of the common peroneal nerve and placing in and about it 30 to 60 mgm. of powdered tetanus toxin. Rigid tetanic extension of the limb followed in about three days. Six, twelve, and seventeen days later no definite change could be observed in the cord or ganglion cells corresponding to the tetanized limb.

The injection of a lethal dose of strychnine (0·83 mgrm.), once repeated, in a decapitated preparation (Sherrington [27], pp. 137-141) produced recurring tonic and clonic spasms over a period of four hours. Subsequent examination of the Golgi apparatus in the spinal cord cells revealed no changes.

If, therefore, it is possible to produce a condition of fatigue in the cells by tetanus or strychnine convulsions, at least there is no associated change apparent in the Golgi apparatus.

SUMMARY OF RESULTS.

Decerebration and high section of the cord are without effect on the Golgi apparatus of the anterior horn cells. Following tetanus, there were no changes in the cord or ganglion cells Administration of strychnine, causing repeated convulsions, likewise gave a negative result. The increased muscular tone of decerebrate rigidity and the muscular flaccidity following cord section must be associated with considerable variation in the activity of the motor cells. Likewise, the constant muscular contraction of tetanus, which is sufficient to cause actual muscular wasting, indicates greatly increased activity on the part of the motor cells. Nevertheless, under the above conditions, I have not been able to discover any change in the Golgi apparatus of the neurones in the spinal cord or ganglia.

Section of the axone is followed by retispersion, a specific alteration of the Golgi reticular apparatus in nerve cells. The complete reaction is made up of three stages: (1) Displacement of the unbroken apparatus to the periphery of the cell and away from the axone-hillock—retispersion (figs. 3 and 5). (2) Dissolution of the reticulum—retisolution (fig. 6); frequently there is very little evidence of retisolution. (3) Reconstruction.

In general, chromatolysis and retispersion are associated, but they may appear independently. Retispersion has been the first to appear in the experiments reported here. In a particular cell, the reticulum may be peripheral or even absent, while the nucleus remains central and the chromophil substance appears normal.

Retispersion appears early in Clarke's column, being far advanced on the fourth day after cord section. The motor cells seem to be more resistant to alteration. The Golgi apparatus appeared at the periphery of a small proportion of these cells on the seventh day and this became quite general on the sixteenth day after cutting the sciatic, but fragmentation was infrequent.

In the spinal ganglia, after section of the peripheral neurone-outgrowth, the fourth day saw the beginning and the seventh day the height of retispersion, with the appearance, also, of considerable retisolution. The cutting of the central division of the axone-outgrowth, the posterior nerve root, induced retispersion in the corresponding ganglia. But section of the cord failed to produce any change in the ganglia below, probably due to the fact that all of the central fibres could not have been thus severed.

To sum up briefly: In the above experiments, no changes were observed in the Golgi apparatus, following variations in nerve cell activity. The apparatus showed no detectable alteration after the removal of the influence of *connecting* neurones, but it exhibited a delicate and specific response, retispersion, to a lesion of its axone-outgrowth, whether central or peripheral. Retispersion is an added feature of the cellular reaction to axone interruption, usually called chromatolysis. It is ordinarily the first and in some cases the only manifestation of this cellular reaction.

In conclusion, it is a pleasure to thank Professor Sherrington for his interest and helpful criticism; also, Mr. H. M. Carleton and Dr. J. B. Gatenby for many useful suggestions.

BIBLIOGRAPHY OF CITED LITERATURE.

[1] BENSLEY, R. R. "On the Nature of the Canalicular Apparatus of Animal Cells," *Biol. Bulletin*, Woods Hole, Mass., 1910, vol. xix, pp. 179-194.

[2] CARLETON, H. M. "Note on Cajal's Formalin-silver Nitrate Impregnation Method for the Golgi Apparatus," *Journ. Roy. Micr. Soc.*, 1919, pp. 321-328.

[3] *Idem.* "Observations on an Intranucleolar Body in Columnar Epithelium Cells of the Intestine." *Quart. Journ. Micr. Sci.*, 1920, vol. lxiv, pp. 329-343.

[4] COLLIN, R., and LUCIEN, M. "Réseau interne de Golgi dans les cellules nerveuses des Mammifères," *Compt. rend. Assoc. des Anat.*, 1909, t. xi, pp. 238-245.

[5] COWDRY, E. V. "The Relations of Mitochondria and other Cytoplasmic Constituents in Spinal Ganglion Cells of the Pigeon," *Internat. Monatschr. f. Anat. u. Physiol.*, 1913, vol. xxix, pp. 473-498.

[6] DUESBERG, J. "Plastosomen, 'Apparato reticolare interno' und Chromidialapparat," *Ergebnisse der Anat. u. Entwickelungsgeschichte*, 1912, Bd. xx, S. 567-916.

[7] *Idem.* "Trophospongien und Golgischer Binnenapparat," *Anat. Anz.*, 1914, Bd. xlvi, Ergänzungsheft, S. 11-80.

[8] GATENBY, J. "The Cytoplasmic Inclusions of the Germ Cells. Part V. The Gametogenesis and Early Development of the *Limnea Stagnalis* (L.), with Special Reference to the Golgi Apparatus and the Mitochondria," *Quart. Journ. Micr. Sci.*, 1919, vol. lxiii, pp. 445-493.

[9] *Idem.* "The Cytoplasmic Inclusions of the Germ Cells. Part VII. The Modern Technique of Cytology," *Quart. Journ. Micr. Sci.*, 1920, vol. lxiv, p. 3.

[10] GOLGI, C. "Intorno alla struttura delle cellule nervose," *Boll. Soc. Med. Chir. di Pavia*, 1898, pp. 53-63; or *idem*, "Sur la structure des cellules nerveuses," *Arch. ital. de Biol.*, 1898, t. xxx, p. 60.

[11] Golgi, C. "Sur une fine particularité de structure de l'épithélium de la muqueuse gastrique et intestinale de quelques vertébrés," *Arch. ital. de Biol.*, 1909, t. li, p. 213; *Arch. per le Sci. Méd.*, 1909, t. xxxiii, p. 1.
(For full bibliography of Golgi's work on this subject, see this article, p 215.)

[12] Holmgren, E. "Studien in der feineren Anatomie der Nervenzellen," *Anat Hefte*, 1900, Bd. xv, S. 1-90.

[13] *Idem.* "Einige Worte über das 'Trophospongium' verschiedener Zellarten," *Anat. Anz.*, 1902, Bd. xx, S. 433-440.

[14] *Idem.* "Trophospongium und Apparato reticolare der spinalen Ganglienzellen," *Anat. Anz.*, 1914, Bd. xlvi, S. 127-138.

[15] Kopsch, F. "Die Darstellung des Binnenetzes in spinalen Ganglienzellen und anderen Korperzellen mittels Osmiumsäure," *Sitzungsber. d.k. preuss. Akad. d. Wiss.*, Berlin, 1902, S. 929.

[16] Legendre, R. "Recherches sur le réseau interne de Golgi des cellules nerveuses des ganglions spinaux," *Anat. Anz.*, 1910, Bd. xxxvi, S. 207-215.

[17] Marcora, F. "Di una fina alterazione delle cellule nervose del nucleo di origine de grande ipoglosso consecutiva allo strappamento ed al taglio del nervo," *Boll. Soc. Med. di Pavia*, 1908, t. xxii, pp. 134-137.

[18] *Idem.* "Ueber die Beziehungen zwischen dem Binnenetze und den Nisslschen Korperchen in den Nervenzellen," *Anat. Anz.*, 1910, Bd. xxxv, S. 65-69.

[19] *Idem.* "Ueber die Histogenese des Zentralnervensystems mit besonderer Rücksicht auf der innere Struktur der Nervenelemente," *Folia Neuro-biol.*, 1911, Bd. v, S. 928-960.

[20] Nissl. "Ueber die Veranderungen der Ganglienzelen am Facialiskern des Kaninchens nach Ausreissung der Nerven.," *Allgem. Zeitschr. f. Psychiatr.*, 1892, Bd. xlviii S. 197-198. (Quoted by Van Gehuchten, 30.)

[21] Nusbaum, J. "Ueber den sogenannten inneren Golgischen Netzapparat und sein Verhaltnis zu den Mitochondrien, Chromidien und anderen Zellstrukturen im Tierreich (Zusammenfassendes Referat)," *Arch. f. Zellforsch.*, 1913, Bd. x, S. 359-367.

[22] Penfield, W. G., 1920. "On the Relationship of the Golgi Apparatus and Holmgren Trophospongium in Nerve Cells. Comparison during Retispersion." (To appear shortly.)

[23] Ramon y Cajal, S. "Estudios sobre la degeneración y regeneración del systema nervioso," 1914, t. ii, Madrid.

[24] *Idem.* "Algunas variaciones fisiológicas y patholǒgicas del aparato reticular de Golgi," *Trab. Lab. Invest. Biol. Univers.* Madrid, 1915, t. xii, p. 2.

[25] *Idem.* "Variaciones fisiológicas del reticulo de Golgi en algunos elementos epiteliales y mesodérmicos," *Bol. de la Soc. Espanola de Biol.*, 1915, March.

[26] Sjövall, E. "Ein Versuch, das Binnennetz von Golgi-Kopsch bei der Spermato und Ovogenese zu homologieren," *Anat. Anz.*, 1906, Bd. xxviii, S. 561-579.

[27] Sherrington, C. S. "Mammalian Physiology," Oxford, 1919.

[28] Sherrington, C. S., and Laslett, E. E. "Remarks on the Dorsal Spino-cerebellar Tract," *Journ. of Physiol.*, 1903, vol. xxix, pp. 189-194.

[29] Van Gehuchten, A. "A propos du phénomène de chromatolyse," *Bull. Acad. roy. de Méd. de Belg.*, 1898, 4me sér., t. xii, pp. 222-230.

[30] *Idem.* "Anatomie du système nerveux de l'homme," 4th edition, Louvain, 1906.

Section of Neurology of the Royal Society of Medicine.

President—Dr. W. Aldren Turner, C.B.

Dr. S. A. Kinnier Wilson read a paper "On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits,"[1] which appears on p. 220 of this number. A discussion followed, in which took part:—

DISCUSSION.

Dr. H. C. **Bazett**: I have found Dr. Kinnier Wilson's paper of great interest since his cases show such striking similarities with the decerebrate animals described by Professor Sherrington, and yet in some respects no less definite contrasts. I have recently been employed in Professor Sherrington's laboratory in developing a technique by which cats may be kept under observation for two or three weeks, even after removal of all nervous tissue lying anterior to the posterior colliculi. These chronic decerebrate animals differ only slightly from the acute preparations, but in some ways they provide a better standard for comparison with clinical material, and their importance lies in the fact that the localization of the lesion can be accurately determined.

The main similarities are that in these animals too the extensor rigidity may be maintained for days or weeks, that this position may alternate with positions of flexion, and that tonic fits with marked opisthotonos may be occasionally seen soon after the operation. A common result is to obtain an animal, which keeps up usually a position of extensor rigidity, but which after a few days also shows occasionally positions of flexion, and this flexion is sometimes flaccid but may be rigidly maintained. It is also common for a preparation, which, when approached quietly (since these animals are often very sensitive to auditory stimuli), is seen to be lying in a flexed position, to assume a position of extensor rigidity as soon as any examination is attempted. In some cases this may be induced by mere auditory stimuli.

The occurrence of tonic fits is comparatively rare, and is limited to the first few hours after the operation, except in some cats in which meningitis has developed. In the early cases the opisthotonos is extreme, the respiration is generally slow but may be fast and shallow, and occasionally with a slow respiratory rate the fits themselves have a respiratory rhythm. These animals are very different from the better preparations, in which the respiratory rate is normal, and they rarely survive long if the fits are at all severe. The operation often causes a certain amount of hæmorrhage below the tentorium into the subarachnoid space, but this hæmorrhage seems to have been particularly noticeable in the animals which have shown tonic fits. In any case the origin of these fits in the nervous system below the corpora quadrigemina is confirmed.

---

[1] At a meeting of the Section, held May 13, 1920.

The main contrasts are that these preparations never show any athetoid movements or tremor, that they never show a pyrexia but a complete loss of temperature control with a rapid fall of body temperature, unless they are artificially kept warm.

They have a temporary glycosuria which disappears within about three days after the operation; they show brisk corneal reflexes, while vocalization in the form of purring, growling, and very rarely mewing may occur.

A few of the animals have survived sufficiently long to allow degeneration to take place, and the remaining parts of the central nervous system are being examined for me by Dr. W. G. Penfield, so that further data should shortly be available. It is at any rate clear that decerebrate rigidity persists even after the rubrospinal tract is mostly degenerated, so that if the red nucleus is responsible for the rigidity it must be the lower part of this nucleus that is concerned: and it is established that the rigidity is not due to the excitation of nerve fibres divided in the operation, since in this case the rigidity could not be maintained after the degeneration of the fibres.

Dr. WALSHE: While we may agree with Dr. Wilson that tonic or postural "fits" are probably of the nature of accentuations of Jackson's cerebellar attitude, I do not think that Jackson's view of the origin and relationship of these phenomena has always been correctly interpreted by those who have at various times referred to his work. In this respect, I notice that Dr. Wilson has adopted the usual view that Jackson regarded both the persisting attitude and the "fits" as identical in origin. This is not what Jackson held, and I have always been puzzled to understand how it was that in this connection he departed from his usual teaching in respect of rigidity and ascribed it in this instance to *cerebral* influx. If we refer to the papers in which he described his cases of cerebellar tumour with "tetanus-like seizures" and cerebellar attitude, which were reprinted in vol. xxix of BRAIN in 1906, we find (p. 432) the statement that "the patient's persisting attitude (cerebellar paralysis with rigidity, that is, a cerebellar attitude). . . . was, in general appearance, like that in the tetanus-like seizures, but the process of causation of the two conditions is, in my opinion, very different." And again (on p. 441), "the rigidity fixing the attitude is the result of cerebral influx, that influence being no longer antagonized, or being less antagonized, by cerebellar influence. Tetanus-like seizures are, I submit, owing to changes of instability set up in nerve cells near the tumour, owing to a cerebellar discharge lesion; the tetanus-like seizures are owing to occasional intense discharges of this persisting discharge lesion." For various reasons it is extremely difficult to believe that phenomena so closely alike in their characters can differ widely in origin, or that the persisting attitude is an expression of cerebral influx. It is far more probable that, whether we accept their identification with decerebrate rigidity or not, both have a common cause, as Dr. Wilson believes. However, it is not correct to attribute this view to Jackson.

PUBLICATIONS RECENTLY RECEIVED.

Cerebrospinal Fever. By MICHAEL FOSTER, M.A., M.D., and J. F. GASKELL, M.A., M.D. Pp. 222. Cambridge University Press, 1916.

In this volume the authors give a systematic account of cerebrospinal fever, based upon their extensive experience in the epidemic of 1914-15. They show reasons for preferring this term to the longer one in common use, epidemic cerebrospinal meningitis. The book deals fully with every aspect of the disease, but is admirably concise. The clinical section is particularly well arranged, and we have seldom read a clinical description at once so complete and so lucid. The chapters devoted to the evolution and course of the malady and its diagnosis are excellent and prepare the reader for the later chapters dealing with the various clinical forms of cerebrospinal fever, so that these do not have the arbitrary and artificial precision so common in clinical classifications.

The authors have attempted, as far as possible, to correlate the clinical manifestations with the underlying pathological lesions. They regard the essence of the disease as a true infection of the cerebrospinal system, causing death in fatal cases by the intensity of the pathological process, and not as a meningococcal septicæmia in which the blood-borne infection is the potent factor.

Both toxæmic and physical factors, however, play a part in the disease process. Thus, they suggest that the purulent exudate overlying the brain acts both by poisoning and by compressing the brain. It is probable, however, that some of their suggestions in this respect have but little foundation. For example, they regard the characteristic head retraction as a manifestation of increased intracranial tension. If this were so, we should expect to find head retraction a common symptom in cerebral tumour, whereas it is of extreme rarity. It is more likely that it is an expression of the extensor rigidity which Jackson described under the name of cerebellar attitude, or it might even be a reflex spasm of the neck extensors from meningeal irritation. The work of Cushing and his school upon the pathways of the cerebrospinal fluid and upon the production and nature of internal hydrocephalus was not known in this country at the time this monograph was written, and it throws light upon several points raised by Foster and Gaskell, and perforce left unanswered by them.

In the chapter devoted to prognosis they note the rarity of residual paralytic phenomena and the absence of mental defect as a sequel of cerebrospinal fever.

They hold that the essence of all treatment lies in the adequate drainage of the subarachnoid space by repeated lumbar punctures, long persisted in in chronic cases. The object of each puncture should be complete evacuation of the excess of fluid, which is always found, except in a few chronic suppurative cases.

The series of coloured plates, at the end of the text, scarcely do justice to this valuable monograph, which must become the standard work of reference on this disease in the language. There is a complete bibliography.

Instinct and the Unconscious. A Contribution to a Biological Theory of the Psycho-neuroses. By W. H. R. RIVERS, M.D., D.Sc., LL.D., F.R.S. Pp. 252. Cambridge: University Press, 1920.

After many years spent in teaching psychology and the physiology of the senses at Cambridge, Dr. Rivers turned his attention to anthropology, and is well known as the author of the "History of Melanesian Society," and for his "Studies on Kinship and Social Organization." After the outbreak of war, he was engaged in treating wounds of peripheral nerves, but soon became attached to Maghull Military Hospital, and later to Craiglockart War Hospital, where he had unrivalled opportunities for gaining experience of the psychoneuroses of war. Later, he was appointed to the staff of the Royal Air Force Hospital at Hampstead; here, and on the aerodrome at Hendon, he accumulated an unique experience of the temperament and neuroses of the flying man.

The results were embodied in a series of papers published during the last three years: these form six appendices at the end of this book, and contain an account of "A Case of Claustrophobia," "The Repression of War Experience," "War Neuroses and Military Training," and an excellent short note on "Wind-up."

The bulk of the work, however, is occupied in an attempt to bring the abnormal phenomena of mental life into harmony with the processes and activities with which we are now familiar on the physiological level. Such subjects as the unconscious, suppression and inhibition, the nature of instinct, the danger instincts and suppression, dissociation, suggestion, sleep, the substitution neuroses, regression and sublimation, are dealt with in a series of short chapters. These are clear and admirably written, and will be of the greatest value to all who are interested in the nature and workings of the unconscious. Moreover, the theory that instincts are suppressed primitive mental activities, which may be either held entirely in check or used in part, is a brilliant addition to our knowledge. Dr. Rivers has much to say on the mechanism of suppression and dissociation, which is equally luminous. It is impossible in a short space to give an adequate account of the originality and interest of this book, which deserves repeated reading.

Injuries of the Peripheral Nerves. By HENRY S. SOUTTAR, C.B.E., F.R.C.S., and EDWARD W. TWINING, M.R.C.S., L.R.C.S. Pp. 152 with 30 figures. Bristol: John Wright, 1920.

This is an admirable book clearly written and excellently illustrated. No attempt is made to enter into theoretical questions, but the gross effects produced by division of each of the larger nerve trunks are considered in detail, with especial relation to the site and nature of the injury. As might be expected from a surgeon of Mr. Souttar's wide experience, the various operative procedures are clearly described with the help of admirable drawings. It is encouraging to find so close an agreement between the conclusions arrived at independently by the authors of this work and the Committee of the Medical Research Council. It shows that, at any rate in England, a harmonious body of opinion is gradually growing up on the diagnosis and treatment of peripheral nerve injuries. We strongly recommend this practical book to all who are interested in the problems of the surgery of nerve lesions.

The Diagnosis and Treatment of Peripheral Nerve Injuries. [Report of the Committee upon Injuries of the Nervous System.] Published by the Medical Research Council. Pp. 59. London: His Majesty's Stationery Office, 1920.

An attempt has been made in this pamphlet to lay down the general principles which should guide diagnosis and treatment in cases of injury to the peripheral nerves. But, in addition, the main nerve trunks are considered individually, and the various difficulties, due to peculiarities in their constitution and function, are considered in each instance. All theoretical questions are avoided and the advice offered, or procedure described, is given in the simplest terms.

Part I deals with nerve injuries in general, treating succinctly of diagnosis, non-operative treatment, the nature of the various operations to be performed, the signs of returning function and the phenomena of pain. In Part II, the signs and symptoms of injury, together with the methods of treatment to be adopted, are considered for each of the main nerve trunks in turn. In Part III the histological appearances are described as they appear under different conditions of injury. Finally, the treatment of painful states, such as "causalgia," and the factors which produce them are dealt with briefly. The Report is intended as a succinct guide for those who are engaged in treating nerve injuries resulting from the war, or in assessing the various degrees of disability they may produce.

Diagnosis and Treatment of Brain Injuries with and without a Fracture of the Skull. By WILLIAM SHARPE, M.D., Professor of Neurologic Surgery, New York Polyclinic Medical School. Pp. 757. Philadelphia and London: Lippincott, 1920.

The author deals with methods of diagnosis and describes in detail the signs and symptoms, treatment and progress of individual patients with various forms of brain injury. He rightly insists on the importance of considering the form and situation of the lesion of the brain, as opposed to the injury of the skull. For, in treating patients with head injuries, the important factor is not the presence or absence of a fracture, but rather the nature of the intracranial damage and increased cerebral tension. Stress is also laid on the necessity for considering the future condition of the patient, as well as preservation of his life.

The book is divided into three main parts:—

(1) General conditions, such as pathology, diagnosis and treatment, including operative technique, (2) Brain injuries in adults, both acute and chronic; (3) Brain injuries in new-born babies and children. Full records are given of 195 cases and the illustrations are good.

It is urged that the enormous mortality, often 50 per cent, following operative treatment in the acute stage, is largely due to the inopportune time chosen by surgeons for interference. The two periods during which operation is distinctly contra-indicated are, firstly, during the early stage after the injury, when the patient is suffering from shock, and, secondly, after signs have appeared of medullary compression. In the author's opinion, expectant palliative treatment is successful in two-thirds of the cases, excluding depressed fracture of the vault; but in the remaining third, where there is considerable increase of intracranial pressure, the skull and dura should be opened, preferably in the subtemporal region. We note with surprise that all methods for closing cranial defects are condemned, and no mention is made of the distressing symptoms which are liable to develop in patients with large openings in the skull.

The Medical Problems of Flying, including Reports I to VII of the Air Medical Investigation Committee. Published by the Medical Research Council. Pp. 272. London: His Majesty's Stationery Office, 1920.

During the course of the war, flying was gradually carried on at greater and greater heights, so that by the autumn of 1916 "mountain sickness" in various degrees gravely increased the difficulty and dangers met by flying officers in the course of their work in the air. Much experimental work was undertaken by individual medical officers, expert physiologists, and civilian physicians, and a series of special reports were published under the ægis of the Medical Research Committee. The papers which comprise this volume contain much that is both directly and indirectly of interest to neurologists. They deal with the reaction of pilots and observers to diminish oxygen pressure, as measured by Professor Dreyer's method, and by the various tests introduced by Colonel Flack. The papers on "Methods used in selecting Candidates for Flying," "On the Sense of Stability and Balance in the Air," on "Mental Aptitude for Aviation," and on "Reaction Time Tests carried out on Flight Cadets," all bear on the functions of the central nervous system in an unexpected and novel manner. Some of the tests described are worthy of more extended application to neurological practice.

Les Maladies nerveuses. Cours professé à l'Université de Louvain. By the late Professor A. VAN GEHUCHTEN. Pp. 645. Louvain: Librairie Universitaire, 1920.

Professor van Gehuchten was preparing to publish the course of lectures delivered by him in the University of Louvain, when the work was stopped by the outbreak of war. The first four hundred pages were already in print, the remainder was in manuscript. The plates prepared to illustrate this book were burned by the Germans, but they have been excellently reproduced from photographs, and a large number of diagrams show the anatomical relations necessary for understanding the signs of the various diseases described. The whole has been passed through the press by the filial piety of Dr. Paul van Gehuchten in memory of his distinguished father.

As the title indicates, this book is intended for students, not for neurologists, and probably for this reason all references are omitted. Organic diseases are classed under such headings as peripheral nerves, muscles, spinal cord, mid-brain, &c. This necessitates somewhat summary treatment, for all morbid conditions are described in terms of anatomy, and little attention is paid to disturbance of function. The last section of the book is devoted to "functional nervous disorders" grouped under the headings of chorea, paralysis agitans, epilepsy, hysteria, neurasthenia and traumatic neuroses. It is a profound misfortune that the lamented author died before his work was completed and without having the opportunity to embody the great advances in neurology made during the last six years.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[JANUARY, 1921.]

BRAIN.

PART 4, VOL. 43.

LEFT-HANDEDNESS AND MIRROR WRITING, ESPECIALLY AMONG DEFECTIVE CHILDREN.

BY HUGH GORDON, M.A.

PAGE
CHAPTER I.—INTRODUCTION 313
CHAPTER II.—TESTS AND METHOD OF TESTING CLASSES 316
§ 1.—Tests 316
(a) Rubbing and Throwing 317
(b) Throwing 318
(c) Scissors Tests 319
(d) Picking-up Test 321
(e) Winding Test 322
(f) Line Test 322
(g) Grip Test 325
(h) Other Tests 325
§ 2.—Method of Testing Classes 326
(a) Mental Defectives and Infants 326
(b) Elder Boys and Girls 327
CHAPTER III.—RESULTS AMONG NORMAL AND MENTALLY DEFECTIVE CHILDREN .. 330
CHAPTER IV.—TWINS AND LEFT-HANDEDNESS 333
CHAPTER V.—MIRROR WRITING 336
CHAPTER VI.—DEFECTIVE SPEECH AND LEFT-HANDEDNESS 362
SUMMARY AND CONCLUSIONS 364

CHAPTER I.—INTRODUCTION.

THE following observations and experiments were made, unless otherwise stated, in special schools (for mentally defective children) in London and Middlesex.

The Elementary Education (Defective and Epileptic Children) Act, 1899, defines the children, for whom these schools are provided, as "not being imbecile and not being merely dull or backward, are defective, that is to say, children, by reason of mental or physical defect, are incapable of receiving proper benefit from the instruction in the ordinary public elementary schools, but are not incapable, by reason of such defect, of receiving benefit from instruction in special classes or schools, &c."

The investigation originated from an observation made in April, 1918, during the inspection of one of these special schools. In one of the classes nine (37 per cent.) of the children out of twenty-four were writing with their left hands, and doing most of their work left-handedly. At other mental defective schools the percentage of left-handed children appeared to be very high; and head teachers frequently reported that there was a large number of left-handed children in their schools. As the percentage of left-handed children was undoubtedly much higher in mental defective than in the ordinary elementary schools, an attempt was made to determine as accurately as possible their number, and to include in this number those who were originally left-handed, but who had been converted into right-handedness in many of their activities by the pressure brought to bear on them by parents or teachers.

A left-handed child was taken to be one who would use the left hand in preference to the right in such activities as are usually carried out by the right hand in the case of right-handed children. It was originally considered important that the tests should be in such things as might have passed unnoticed by parents and teachers, and for this reason might have remained unchanged. Owing to little time being available at first for the investigation, it was necessary that the tests should be of the simplest character and that they could be applied rapidly, and to whole classes. A great variety of experiments have been made, and only those tests which gave consistent results have been retained. The methods used and the results obtained will be given later.

The high percentage of left-handed children in mental defective schools does not appear to have received any special attention. The following references, however, are of interest in connection with the subject.

Ballard in an investigation on "Sinistrality and Speech" in 1912, notices a higher percentage of left-handed children in mental defective schools than in ordinary elementary schools, i.e., 6·5 per cent. against 4·1 per cent.[1] In both percentages he includes sinistrals (left-handed writers, &c.), and what he calls dextro-sinistrals (left-handed children who write with the right hand).

Smedley [28], 1899, in some experiments on the "strength of grip" of right and left hand concludes that "dull pupils are more ambidextrous than average, and average than bright pupils; while the John Worthy school boys (incorrigibles, truants, &c., Chicago), are more ambidextrous than the dull pupils of the regular schools."

---

[1] These numbers were obtained from reports, &c., from the schools. In his own investigations he gives the percentage in ordinary schools as 2·8 per cent.

In other experiments on children, e.g., in the "tapping tests" (i.e., tapping as rapidly as possible with the right and with the left hand in a given time), Smedley again comes to the same conclusion: "left hand efficiency nearly approaches right hand efficiency in the case of dull and backward children." This conclusion, however, is not confirmed by Bolton. It must not, however, be forgotten that, in the case of left-handed children and adults who write with the right hand, the difference between the efficiency of the hands is likely to be less than when a person is simply right-handed or left-handed. In fact, during the present investigation there appeared to be little evidence of real ambidexterity; there were a few exceptional cases in which either hand was used indiscriminately. The children were of very low-grade type, and their lack of so-called "dexterity" was very marked in the case of both hands.

In "Ambidexterity," J. Jackson [13] refers to the opinion that the insane have a great percentage of "partial ambidexters" in their ranks, and that there is a high percentage of left-handed among criminals; also that left-handed children have been looked upon as somewhat daft, hence nurses and parents did their best to make them right-handed.

Lombroso, however, states that the percentage of left-handed among lunatics does not greatly differ from that among normal people, but that it is much higher among criminals (*North American Review*, 1903). Mayet (1902) found a high percentage (16 to 30) of ambidextrous and left-handed among idiots and epileptics. Lombroso himself considered left-handedness as a sign of degeneracy.

Ballard in 1912 [5] and W. Franklin Jones [14] in 1918 both came to the conclusion that stammering in a high percentage of cases is closely associated with the "transfer" of congenital left-handers to right-handedness in writing and vice versa.

General attitude of teachers towards left-handed children.—In mental defective schools, teachers as a rule after some attempts at converting left-handed children into right-hand writers, and if these attempts are not very successful, leave them to their own devices. Knitting and sewing however are treated differently in the majority of cases, owing to the difficulty experienced by teachers in teaching these subjects left-handedly. As a matter of fact very few were found to knit or sew left-handedly. Probably the lack of adaptability of mentally defective children renders it extremely difficult to bring about these changes (especially in writing); and this may explain why teachers in these schools have found it advisable to leave the children alone. In one school the teachers try to make all the scholars right-hand writers. In this school twenty-eight (46 per cent.) out of a total of sixty-one were found to be left-handed by the various tests described later. Of these twenty-eight, sixteen were well known to have written or to have drawn with their left hand at one time or other. In connection with this point it must be remembered that few children are sent to these special schools before the age of eight, and it is therefore highly probable that most of them have experienced some pressure to make them write with their right hand. Clearly also the pressure had been insufficient to bring about the change. Of these twenty-eight, thirteen are reported to have some "speech difficulties," and two to be "defective in speech," one to "stutter," and two to "stammer" at times. Too much stress must not be laid on such defectiveness, as difficulties in speech apart from left-handedness are not uncommon among this class of children. At another school the children are especially trained in ambidexterity and even in writing with both hands.

In ordinary elementary schools, teachers generally assert that at the present time little or no pressure is brought to bear on left-handed children. As a matter of fact most left-handed children are subjected to some pressure either by teachers or parents, but generally unaccompanied by any harshness. A great many of these left-handed children are of a highly nervous temperament, and in many cases this nervousness does not appear to be due to a forced change of hands, or to an unsuccessful attempt to convert them into right-hand writers. It is however difficult to say anything very positively, as it is almost impossible to ascertain the true facts of each case.

CHAPTER II.—TESTS AND METHODS OF EXAMINATION.

§ 1.—*Tests.*

A great many experiments have been made, e.g., kicking, tying knots, the use of the hammer and saw, &c., but in most of these many difficulties were encountered, and in some it was found that the results were inconsistent with the known facts. In kicking a ball for example, it was often found that thoroughly left-handed boys kicked with the right foot, and in the case of girls and younger children no reliance could be placed on the results, as they frequently kicked with the foot which happened to be in the most convenient position when they reached the ball.

Originally all children, who could write their names, were asked to make the attempt with their left hand, as it was expected that the result might indicate those who had some skill with this hand. The instructions were very simple ; the children were told to take their pencils in their left hand and to hold them up. In this way it could be seen that everyone had them in the left hand. They were then asked to write their names with that hand, on a slip of paper that had been given them. After a little hesitation nearly everyone made the attempt. It was interesting to note the difficulties they experienced, and how they overcame these difficulties. Many began by holding the pencil four or five inches from the pointed end and attempted to write with an arm, instead of a wrist or finger movement. As a rule however they gradually changed to a more suitable position and movement. It was remarkable—considering the class of children—how well they wrote their names as a rule. One peculiarity was immediatey noticed —namely "mirror writing." As this type of writing was found to be prevalent among mental defectives, a special investigation was made into the matter, the results of which are given separately.

After prolonged experiments the following tests were found to be the most reliable and most consistent with known facts. In fact very seldom was a known left-handed child missed, and very frequently left-handed children, unknown to the teacher, were discovered who on careful inquiry were found to have been left-handed in their earlier school career.

(*a*) *Rubbing and throwing test.*—The names of the children in a class were written on a sheet of foolscap and the experimenter went round to each child with a duster and placed it on the desk, asking the child to give the desk a good rub. As a rule the child seized the duster, and rubbed the desk vigorously with the right hand ; the left-handed child, however, seized the duster and rubbed the desk with the left hand. It was very necessary to look at the non-rubbing hand, as not infrequently a right-handed child with a penny or handkerchief in the right hand rubbed with the left hand. Those who rubbed with the left hand were immediately asked to stand at the back of the room, and to catch the folded-up duster and throw it back. In infant schools these two tests were remarkably successful. The test was very quickly carried out; and throwing with the left was found very often to be associated with left-hand rubbing. The teacher, who had the list of names, entered "L" in the columns headed "Rub" and "Throw" in the case of those who did it with their left hand. When a child rubbed with the left hand and threw with the right, he was again asked to rub the desk, care being taken to place the duster so that it could be taken more easily with the right. If he still persisted in rubbing with the left the letter "L" was entered, but, if he now changed to the right, the letter was entered but a line was drawn through it. Interesting cases were continually met with when a child was asked to catch the duster; some immediately placed the left hand behind their backs or held their left arm stiffly down with their fist clenched and put the other hand well in front. Some of these actually rolled up the duster with the right hand, still holding the left arm down or behind them. It was evident they were not acting in a natural way, that they were in fact inhibiting the natural action.

Among the older children in ordinary schools, i.e., those above Standard II (about 8½ years of age) and in the elder boys and girls, special schools (over 12), the rubbing and throwing tests were not so successful for various reasons. Often those who were being tested soon understood the object of the test; some wished to conceal their left-handedness, others thought that the trial was to find out those who could throw well with the left hand. It is important in these and the following test that both should be made before any knowledge of their exact purpose has been gained.

As an example of the result of these two tests the following is given:—

Infant school, the lowest class, ages 4½ to 5½, forty-one boys and girls. Seven were found to rub and throw with the left hand and one to rub with the left but throw with the right. Three days afterwards the whole class were asked to draw anything they liked, and the seven drew with the left hand, and all the others with the right.

(*b*) *Throwing tests.*—A test for older boys and girls. A waste-paper basket was placed about four or five yards from a line marked on the floor. After explaining that it was wished to discover those who could throw the straightest, by seeing who could throw a knotted duster into the basket from the line marked on the floor, the experimenter sat at the table with the list and called out in order the names of the boys or girls, and noted those who threw with the left hand. In this way the test was carried out without the "subjects" having any knowledge of the real object of the experiment.

The results of this test in three elder boys' schools (for mentally defective) are given below :—

School A —Classes 1 and 2: thirty-one boys, ages 13 years to 16 years 8 months. Five boys (and five boys only) threw with the left hand; all of these and no others were conclusively proved to be left-handed. Of these, two always write with the left hand, and one with the right; the remaining two write with the right, but used to write at one time with the left.

School B.—Classes 1 and 2: forty-six boys. Four boys threw with the left, of these three were proved to be left-handed, and they write with the left hand. One had some clear indications of left-handedness, but writes with the right hand. Of the rest, three who threw with the right, each had some slight indications of left-handedness, but insufficient to be noted as left-handed; they all write with the right hand.

School C.—Classes 1 and 2: sixty-one boys, ages 11 years 9 months to 15 years 10 months. Six boys threw with the left; of these five were proved to be left-handed: four of them write with the left and one now writes with the right, but used to write with the left at one time. Of the remainder, two boys who threw with the right, one was clearly left-handed; he draws and writes with the left hand. one had some indications of left-handedness, and is much stronger with the left-hand grip; dynamometer, left 59, right 45.

From the above facts it is evident that left-hand throwing is as a rule a sign of general left-handedness. It is not unlikely however that there may be exceptional cases, where this diagnosis may not be correct, e.g., injury to the right hand or arm at a critical age. On the other hand it is equally clear that throwing with the right hand is not always associated with right-handedness. Parents do undoubtedly effect the change, often at an early age.

(*c*) *Scissors test.*—The scissors in general use are made for right-handed people. In attempting to cut with the left hand anyone naturally holds them in the same way as he has learnt to do with the right hand, and if the two blades are loosely attached these are in consequence forced apart instead of together. The cutting of paper, linen, &c., is impossible under these conditions, especially if the scissors are not very sharp. This test seems to be a most reliable one with children, and especially with dull and backward ones. With adults however, it is different, as a great many have learnt to cut their nails with the scissors in their left hand. Many do not clearly understand how they do it, and a fairly high percentage cannot do it at all. It is often very difficult to teach them, and frequently after having learnt they find they have lost the knack, by merely cutting a piece of paper again with the right hand. Some left-handed children however can cut a piece of paper only with their right hand with these scissors; but as a rule they can cut with the left or with both.

Children who can cut with these scissors with the left hand may be divided into two classes:—

(1) Those who can cut equally well with left and right hands.

(2) Those who can cut with the left but not with the right.

Among the youngest children—about 5 years of age in the ordinary elementary school, and about 7 to 8 in the mental defective school, there are many who cannot cut with these scissors with either hand, but are quite capable of doing so with the ordinary scissors in use in the schools.

A great many tests have been made with these specially prepared scissors, and the following results indicate their reliability. The scissors are merely loosened, and the cutting edge rendered less sharp by filing.

In the case of 318 children in mental defective schools, who cut with the left hand—

(i) 203 cut with left and right; of these, 172 were certainly left-handed (about 86 per cent.), eight had other indications of left-handedness, twenty appeared to be right-handed, and one appeared to be ambidextrous; the remaining two had been taught at home.

(ii) Fourteen cut with the left and could cut a little with the right; of these, fourteen were left-handed (100 per cent.).

(iii) 101 cut with the left only; of these, ninety-seven were left-handed (about 97 per cent.), two were probably left-handed, and two appeared to be right-handed, and no explanation of their skill was found.

Put in another way, out of 318 children who cut with the left hand 283 (89 per cent.) were left-handed, and nine (2·8 per cent.) were probably left-handed.

Below is given the percentage of left-handed children who cut with the left only, or with left and right hands:—

Number of left-handed children		Cut with left and right		Cut with left only
244	.. ..	102 (41·8 per cent.)	..	78 (32 per cent.)

i.e., about 74 per cent. of the left-handed could cut with the left hand, and roughly speaking about a quarter could not cut with the left hand. Only very young right-handed children are unable to cut with these scissors with the right hand. All left-handed children, therefore, are not able with these scissors to cut with their left hand; in fact, only 75 per cent. can do so.

From these facts it is evident that (i) if a child can cut with these scissors with the left hand, and only with the left hand, he is nearly certain to be left-handed, i.e., to use the left hand as the dominant hand in many of his activities.

(ii) If a child can cut equally well with both hands, he is probably left-handed.

(iii) If a child cannot cut with his left but can cut with his right hand, it must not be inferred without further evidence that he is not left-handed in many of his activities.

Attempts were made to discover how some of the markedly right-handed children had learnt to cut equally well with their left hand. In many cases no explanation could be found, and this is what might have been expected, as it is often difficult to obtain information from children found in mental defective schools. The following cases are interesting and are not unimportant when the whole question of left-handedness comes to be considered later on in this paper.

One boy explained that he never could cut with his right, and that his brother, who always cuts with his left and is left-handed, taught him. This boy is still unable to cut with his right hand, as he holds the scissors exactly in the same way as he does with the left, and so forces the two blades apart. In two cases the children who could cut equally well with either hand had been taught at home.

In elementary schools: (i) A girl, aged about 11 years, cut with both left and right hands, but asserted she had never been left-handed. On inquiry the head teacher of the infant school from which she came remembered quite distinctly that at first she used to write with her left hand; (ii) a girl, aged about 7 years, cut equally well with either hand, but in every other test gave no indication of left-handedness. It was then remembered that the girl had broken her right arm some time ago, and had come to school for three months with the arm in a sling. She was found to be able to write her name equally well with either hand.

(*d*) *Picking-up test.*—A large number of left-handed children pick up anything on the ground with their left hand, provided their attention is concentrated on the picking up and not on the question of the hand they are going to use.

Those to be tested were arranged in a line and a small piece of paper was placed exactly in front of each. They were told that there was to be a race to see who could pick up the paper first and hold it up.

It was found best to say: "Stand up straight with your arms down," and immediately afterwards, "Go." In many cases it was noticed that some left-handed children, when getting ready, placed their left hand behind them, apparently to force themselves to pick up with the right. If however they were made to stand up straight with arms held down and started immediately, the left-handed picked it up quite naturally, and with the left hand. Sometimes children started to pick up with the left, and then suddenly changed to the right. Whole classes have been tested; about ten or so were tested at a time in the hall, and those who were holding the paper in the left hand kept back; these, when all the class had been tested, were each given a piece of paper and asked to place it on the floor, and they were then raced again. Under these conditions it was found that right-handed children did not often pick it up with the left, but that in a certain number of cases the left-handed picked it up with the right. The test by itself may not be of much value, but taken with the other tests it is by no means unconvincing.

(*e*) *Winding test.*—As a rule in winding cotton on a reel, one hand is used to hold the reel and the other to wind. Among right-handed people the winding is done by the right hand, and in the case of the left-handed by the left. Sometimes the winding is carried on by the hand holding the reel, in either case however the winding is done by the dominant hand. A few children and a good many adults move both hands while winding the cotton. In such cases a simple apparatus can be used to prevent the movement of the holding hand, and the time can then be taken for winding a yard of string by each hand by means of a stop-watch.

The following experiments show a much greater speed with one hand than the other: a left-handed boy, right = 5 seconds, left = 3 seconds; a right-handed boy, right = 5 seconds, left = 7 seconds.

One fact is very noticeable—the winding by the dominant hand is done chiefly by a wrist movement, whereas when done by the non-dominant hand, the action is from the elbow or even shoulder, i.e., an arm movement. Notice has already been called to a similar movement in writing with the non-dominant hand, and further reference will be made to similar peculiarities in the "line test." It appears therefore that the tendency of the uneducated and unpractised hand is that of an arm and not of a wrist and finger movement.

(*f*) *Line test.*—This is a quantitative test, but it was intended merely to give an indication of left-handedness, especially in cases of doubt. It can be carried out very rapidly and a whole class of even sixty can be tested at the same time.

Slips of paper were distributed to the class, on which to practise making as quickly as possible a number of separate vertical lines of about ¼ to ½ inch in length, first with the right and then with the left hand. The blackboard was used to demonstrate exactly what was to be done. After one or two minutes' practice, new slips of paper were given out, and it was explained that there was to be a race to discover who could draw the greatest number of lines in a given time. The children were told to write their names on the slips, and then to hold up their pencils in their right hand. It was explained that at the word "go" they were to start making lines as quickly as possible and to continue until stopped after fifteen seconds. At the end of fifteen seconds the following instructions were given rapidly: "Stop; put down your pencils, and turn over the paper." Exactly the same instructions were given in the test with the left hand. The test always aroused interest and amusement, especially when the left hand had to be used. The same procedure was used in mental defective and in ordinary elementary schools.

The results were often very convincing, but it was always necessary when the test indicated left-handedness to scrutinize carefully the lines made by each hand, as sometimes left-hand speed was clearly due to less care being taken in drawing the lines.

In some cases, however, especially among children in mental defective schools, no reliance could be placed on the results; many of these children were in fact quite unable to concentrate their attention even for five seconds; and after making a few lines these stopped and looked round to see how the others were getting on. It was interesting to see how nearly every one was anxious to do his best and show his skill. As has already been mentioned, the arm movement of the non-dominant hand was very noticeable. The test was found to be most useful among ordinary children of about 11 years of age and upwards, and among adults.

There was a great variability in the number of lines drawn, but it was evident that as a rule the older and more intelligent the child the greater was the number of lines, as might naturally have been expected. The proportion between the number of lines made by the right and by the left hand varied in every possible way, but in right-handed adults the number made by the right was always at least 29 per cent. more than that made by the left hand. Left-handed adults, who have from childhood written with the right hand, as a rule did about 6 per cent. more with the right than the left.

The results of some of these tests are given in percentages of the number of lines made by the right hand over that made by the left. If a greater number is made by the left hand the percentage is marked by a minus sign, e.g., 50 per cent. indicates that the number of lines drawn in fifteen seconds by the right hand was 50 per cent. more than the number drawn by the left hand, and 50 per cent. (–) indicates that the number drawn by the left hand was 50 per cent. more than that drawn by the right. A higher percentage would therefore point to greater difference in "handedness."

(i) Line test—percentages for right-handed adults: 29 (two cases); 30 (two); 32; 36 (four); 39; 41; 43; 48; 51; 59; 68; 85; 96.

Note.—There was also a case of 21 per cent., but the "subject," who was very right-handed, had broken her right wrist a year ago, thus no doubt lowering the speed of this hand. Another teacher was noted as 80 per cent., and she had always considered herself ambidextrous; she had, however, quite lately injured her left thumb, which would account for a decreased speed with her left hand. A good many experiments have been made with other adults, and in no case, except those given below, was the percentage less than 29 per cent. for the right-handed.

(ii) Line test: percentages for left-handed adults, and for doubtful cases.—

(*a*) Known to be left-handed, but always write with right hand: 21; 13; 7; 6; 5; 4; 1; 0 (two cases); 2 (–).

(*b*) Doubtful cases:—

Twenty-three per cent. Thinks she is right-handed, but in dressmaking always cuts on the left side with left hand; draws equally well with left or right hand; always ties left-handedly.

Fifteen per cent. Does many things left-handedly; has always thought herself ambidextrous.

Five per cent. Did not know he was left-handed; always deals cards left-handedly; throws with left or right equally well.

Five per cent. Not known to be left-handed, but in boxing equally strong with left or right; kicks equally well with either foot; grip dynamometer: right hand 107 kilos., left 105 kilos.

Two per cent. Carves and draws equally well with left or right; left arm (ulna) an inch longer than right; had once suffered from injury to right hemisphere with temporary loss of use of left hand and leg, but with no speech defects.

The average number of lines in thirty-six cases was for the right hand 68·5, for the left 56·5. The greatest number for right hand 88, for left 87; the least number for right 39, for left 21.

(*g*) *Grip test.*—This is a well-known test to measure by means of a dynamometer the strength of grip of the right and of the left hand; a great many observations have been recorded by various authorities. The norms are given for boys and for girls at different ages in the "Manual of Mental and Physical Tests," by G. M. Whipple. The index of right-handedness, i.e., the percentage of strength of the left hand compared with that of the right, was found to range, for an ordinary group of school children, between 91 and 96 per cent. "However, occasionally right-handed children may have an index of strength exceeding 1·00, i.e., grip may be stronger with their left hand. According to Ardlicka, this tendency to have an index contrary to expectation is peculiarly evident in left-handed persons, so that he estimates that nearly one-half of bona-fide left-handed persons may have a stronger grip with the right hand."

The following quotation from the same book is of special interest in connection with the present investigation. "It is often asserted that degenerates tend to be left-handed. Wallin's averages for epileptics show a net preponderance of 0·5 kg. for general average in favour of the left hand, though by no means all his S.'s had an index above 1·00."

It was thought that the grip dynamometer would be very useful in diagnosing left-handedness, but the results were so varying and often so inconsistent with known facts, that little value could be placed on the results obtained by it, unless these were fully supported by other tests. No doubt its inefficiency is due in part to the instrument used, as it is ill adapted to the varying sizes of the children's hands and could not be adjusted. Also there was not sufficient time to allow each child to have as a rule more than one grip with each hand. The dynamometer was taken round to each child, and placed in his hand in the best position and he was told to squeeze as hard as he could. The result was then called out and recorded by the teacher. In this way every one was stimulated to do his best. As a matter of fact every child was eager to make the trial, and wished to try over and over again to improve his record. One curious result was noticed, namely, that in the first trial the two hand grips often approached very closely, not only among mental defectives, but also among normal children and even among adults. After practice the difference between the two hands becomes more marked, but even then it is by no means constant.

Some of the results are recorded in the tables of tests at different schools, but the results must be regarded with a good deal of suspicion.

(*h*) *Other tests.*—Many of the following tests have been tried, but not very extensively. Generally they gave results agreeing with those obtained by the other tests, but only single individuals and those as a rule who were left-handed (or doubtful) were experimented with.

(i) Placing counters in a box one by one, two children being raced to see who could do it the quicker. As a rule it was done by the dominant hand.

(ii) Tying a knot in a piece of string: this was found to give very contradictory results, especially by those who had done "string" work at school.

(iii) Hammering nails and sawing wood, among the elder boys: if done left-handedly the boy was nearly always left-handed.

Taking the tests as a whole, the most unsatisfactory in the case of children in mental defective schools were those quantitative in character, and requiring attention and concentration. The tests which gave the most consistent and reliable results were those in which actions that had apparently become fixed by habit at some time in their early childhood were tested.

§ 2.—*Method of Testing Classes.*

(*a*) *In junior mental defective schools in infant schools and in Standards I and II of the ordinary schools.*—A list of children with their ages is given to the teacher, and each child is tested with the duster as has already been described. The teacher notes those who rub and throw with the left hand. Only those who rub with the left hand are at this stage tested in throwing. Each child is then tested with the scissors, and those who cut with the left are also tested with the right hand, the results being noted by the teacher. Any child found to cut with the left hand is also tested in throwing, if he has not been previously tested, and the result entered as left or right. Sometimes the dynamometer grip test is then tried; at other times the "line" test. Finally those indicated as left-handed in anything are raced in "picking up" and in "winding."

The teachers are then, and not till then, asked to give the names of those who write with the left hand, who used to write or who wished to write with the left hand; also those who draw with the left hand. If any of these have been missed they are tested in throwing, &c. Often in the case of very young children they are asked to draw something on their millboards, and a note made of the hand used.

Some examples of classes tested in this way are given in the tables.

(*b*) *In elder boys' and girls' schools.*—A list of children with their ages is made as in the infant schools, and the teacher records the results. The first test is the "throwing" test, which is followed by the "line" test. In Standards V, VI, and VII this latter test takes very little time, as after the lines have been drawn the children are asked to count the number made by each hand and enter it on the slip. The teacher then calls out each name, and the children read out the number for the right and for the left hand. The slips are collected for scrutiny and verification; they are especially examined in those cases where the number of lines approach each other, or where the lines made by the left hand are more numerous than those made by the right. Each child is then tested with the scissors. Those who appear to have indications of left-handedness are further tested with the "picking up," and "winding" tests. Sometimes the dynamometer is used in addition to, or in place of, the "line" test. Information as to the hand used in writing is then obtained. Examples of the results recorded in classes for older children are given in the tables.

The tests which have just been described appear to be more suitable for children in mental defective schools than quantitative tests (e.g., as tapping, &c.),[1] as the latter involve attention and concentration, both of which are difficult to obtain from these children. In addition, much time and care is required in using them, and the children's skilfulness is only proved in one or two special kinds of work. The qualitative tests, on the other hand, reveal much of the past history of the child, and give evidence of habits, natural or acquired. As a matter of fact, very few were missed who were known to be left-handed, or perhaps it would be better to say, who were known to write or to have written at one time or another with the left hand. In many cases left-handed children were discovered who were not known by their teachers to be left-handed, but who on inquiry were proved to have been left-handed at school or at home. One thing also became clear, namely, that there were all grades of left-handedness. There were some indeed who did everything left-handedly; others who did most things left-handedly, and again others who were right-handed in most of their activities, but had some clearly marked left-handed habits.

At first sight these conclusions may seem to depreciate the value of the tests, but it will be found that the results of the tests are by

---

[1] "Shot tube" test, that is, dropping steel balls into a vertical glass tube, and comparing times taken by each hand. "Pegboard," in which pegs are fitted into holes, and the times taken by each hand compared.

Table I.—School, Mental Defective: Class 1.

Names	Ages	Rub	Throw	Cut	Pick up	Wind	Write	
Boys—	Yrs. mths.							
1	12 0	..	..	..	..	..	..	..
2	14 3	L.	L.	L.R.	R.	L.	R.	Always cuts with left at school
3	10 1	..	..	..	..	..	..	..
4	12 1	..	..	..	..	..	..	..
5	9 9	L.	R.	L.	R.	L	R.(L.)	..
6	11 3	..	..	..	..	..	..	..
7	11 10	L.	L.	L.R.	L.	L.	L.	..
8	12 0	..	..	..	..	..	..	..
9	11 5	..	..	..	..	..	..	..
10	11 4	..	..	..	..	..	..	..
11	11 4	..	..	..	..	..	..	..
12	12 6	..	..	..	..	..	..	..
13	11 5	..	..	..	..	..	..	..
14	11 2	..	..	..	..	..	..	..
15	11 0	..	..	..	..	..	..	..
16	9 5	L.	L.	L.	R.	L.	R.(L.)	..
17	11 4	..	..	..	..	..	..	..
Girls—								
1	12 3	L.	L.	L.R.	L.	L.	R.(L.)	..
2	14 0	..	..	..	..	..	..	..
3	9 5	..	..	..	..	..	..	..
4	9 5	L.	L.	0*	L.	?	R.(L.)	..
5	9 9	..	..	..	..	..	..	..
6	9 3	L.	R.	0*	L.	?	R.(L.)	..

(1) All were tested in rubbing and in cutting, only those indicated as left-handed were further tested in "picking up" and "winding."

(2) All were tested in left-hand writing; no mirror writers.

(3) Under "write" are given the hand habitually used in writing: (L.) indicates that these children were known at the school to have written, or to have wished to write, left-handedly at some period of their school life.

(4) Under "wind" ? indicates that both hands were used at the same time, and that it was therefore impossible to decide which was the dominant hand.

(5) Under "cut" L. = could cut with left *only;* L.R. = could cut equally well with left and right; L.(R.) could cut with left and not at all well with right.

(6) * = not tested in this, e.g., right-hand cutting.

Table II.—School, Mental Defective: Class 2.

Names	Ages	Rub	Throw	Cut	Pick up	Wind	Write	
Boys—	Yrs. mths.							
1	8 11	..	..	..	..	..	..	..
2	10 5	..	..	..	..	..	..	..
3	9 7	..	..	..	..	..	..	..
4	9 10	..	..	..	..	..	..	..
5	10 6	L.	L.	L.	L.	L.	L.R.	Speech indistinct; *has* written mirror.
6	8 8	L.	L	L.R.	L.	L.	L.	Decided epicanthus; *has* written mirror
7	7 11	..	..	..	..	..	..	..
8	7 10	..	..	..	..	..	..	..
Girls—								
1	10 8	L.	L.	L.	L.	L.	L.	Defect of articulation; *has* written mirror
2	9 7	..	..	..	..	..	..	
3	8 3	L.	L.	R.	L.	R.	L.	Very backward and babyish
4	12 7	..	..	..	..	..	..	..
5	8 4	..	..	..	..	..	..	..
6	10 7	..	..	..	..	..	..	..

no means unimportant, when the reason for the larger number of left-handed children in mental defective schools comes under consideration.

In deciding whether a child should be classed as left-handed—in estimating percentages—the following tests as a rule were taken as sufficient:—

(1) Persistent rubbing and throwing with the left hand, especially among very young children; these nearly always also wrote with the left hand.

(2) Throwing and cutting with the left hand.

(3) Writing or having persistently written at one time with the left hand.

TABLE III.—SCHOOL—MENTAL DEFECTIVE, ELDER BOYS: CLASS 2.

Names	Ages		Throw	Scissors	Dynam.		Pick up	Wind	Write	
					L.	R.				
Boys—	Yrs.	mths.								
1	14	4	..	..	40	49	..	..	..	..
2	13	1	..	..	66	75	..	..	..	Best boy but difficult to control
3	14	1	..	..	50	51	..	..	..	..
4	14	3	..	..	10	13	..	..	..	Fairly intelligent. Attending hospital
5	14	7	L.	L.R.	47	54	L.	L.	R.(L.)	Fairly intelligent
6	14	11	..	..	41	28	..	..	..	..
7	14	2	..	..	43	47	..	..	..	..
8	13	9	L.	L.	45	36	L.	L.	L.	Fairly intelligent
9	13	9	..	..	44	40	..	..	..	..
10	13	10	..	..	48	40	..	..	..	..
11	15	1	..	..	35	35	..	..	..	..
12	13	8	..	..	31	29	..	..	..	..
13	13	7	..	..	18	20	..	..	..	..
14	15	11	..	..	30	31	..	..	..	..
15	14	0	..	..	34	39	..	..	..	..
16	13	4	..	..	40	38	..	..	..	..
17	14	3	..	..	33	33	..	..	..	..
18	14	5	..	..	24	30	..	..	..	..
19	16	8	..	..	49	55	..	..	..	..
20	15	3	R.	O.*	38	38	R.	?	R.	*Mirror* writer; very slow. Chorea at one time

Note.—All tested with scissors, throwing and dynamometer; in the other tests only those indicated were tested. All tested in left-hand writing.

Children who could cut with the left hand, but displayed no other left-handed activities, were classed as right-handed. In many cases, however, further inquiries were made into their past history. Children who merely "pick up" or "wind" or do both, were not included among the left-handed. The dynamometer and "line" tests were only used as confirmatory tests. In the great majority of cases, especially in mental defective schools, there was little or no difficulty in coming to a decision. In doubtful cases the children were classified as right-handed.

When it appeared that the percentage of left-handed children in mental defective schools was very much higher than had been found by various authorities among normal children and adults (4 to 6 per cent., and sometimes higher), it became necessary to apply the same tests to the children in the ordinary elementary schools. The same methods were used—one set of tests for children up to about 9, and the other set for older children. As will be seen by the results, the percentage was very much lower than that in the special schools, and somewhat higher than had been estimated by the majority of authorities.

CHAPTER III.—RESULTS AMONG NORMAL AND MENTALLY DEFECTIVE CHILDREN.

Among normal people, Ogle found 4½ per cent. left-handed in 2,000 cases; G. M. Gould gives 6 per cent.; Franklin Jones, 4 per cent.; H. Macnaughton Jones says: "Ghent University had 22 per cent. left-handed (100 cases), but generally the left-handed are estimated at 1 per cent. to 2 per cent."[1] Malgaigne (1859) 8 per cent. to 10 per cent.; Hecht and Langstein (1900), even 12 per cent.; Masini (1907), 10 per cent.; Songues (1,080 cases), 7 per cent., left-handed and ambidextrous; Moutier, at least 10 per cent.

Ballard, in "Sinistrality and Speech" (1912), gives 4·1 per cent. as the result of data obtained from the teachers in reference to children in elementary schools in London. The number of children dealt with was 13,189, and the ages between 4 and 14 years. This percentage is considerably lower than that (7·3) found in elementary schools in Middlesex and London during the present investigation; the number, however, actually tested was only 3,298, and the ages between 4 and 14 years. In his own personal inquiry, however, Ballard reduced the percentage to 2·8; 11,393 cases, and ages between 8 and 14 years. This reduction is remarkable. It seems possible that the omission of the infant schools may have been the cause of the difference in percentages. It is possible that left-handed children may have been over-

---

[1] Josefa Ioteyko, late of Brussels University, states (1917) that there are a considerable number of left-handed in Belgium much above the general estimate of 2 to 3 per cent. [10]

Results of tests:—

Schools	Number tested	Number found to be left-handed	Percentage
(1) Elementary schools, ages 4 to 14	3,298	241	7·3
(2) Mental defective schools, ages 8 to 16	4,620	843	18·2

There appears, therefore, to be more than two and a half times as many left-handed children in mentally defective schools as in ordinary schools.

To discover whether there was any great difference in the percentages of left-handed boys and girls in these special schools, 729 cases were taken haphazard with the following result:—

PERCENTAGE OF LEFT-HANDED BOYS AND GIRLS.

	Number tested	Number found to be left-handed	Percentage	Percentage boys and girls
Boys	374	61	16·3	18·7
Girls	355	76	20·7	

These numbers are not very large, but they appear to represent about the proportion generally found. The proportion of girls to boys as found above is therefore as five to four, in other words the left-handed girls are about 25 per cent. more numerous than the boys. J. W. W. Wallin (1915-16) [26] found by questionnaire the percentage of left-handed among boys 3·6 and among girls 2·1. The number of pupils dealt with was 89,057.

Whether the above percentages are reliable or whether they are too high, one thing is clear to anyone visiting both special and ordinary schools, namely, that there is a very much higher percentage of left-handed children in the special schools, no matter what method of estimating the number is used, whether by merely noting those who write and work left-handedly, or by testing to discover in addition those who were at any time left-handed and have been converted into right-handedness in many of their activities, such as writing, throwing, sewing, knitting, &c.

Some very high percentages were found in some of the classes and in whole schools, as is shown by the following percentages of left-handed children in certain classes, and in whole schools (mental defective).

53 per cent. (47); 45 per cent. (19); 44 per cent. (61); 33 per cent. (27) (39); 31 per cent. (19); 29 per cent. (17). For whole schools: 2 schools over 30 per cent.; 5 schools 25 to 30 per cent.; 13 schools 20 to 25 per cent.; 12 schools 15 to 20 per cent., &c.

The numbers in brackets are the actual numbers tested. In no case was a class specially selected.

In ordinary schools the percentages varied very considerably in different classes. Sometimes the whole school was tested, but generally a few classes were chosen haphazard. The percentages were as a rule between 4 and 10 per cent. In three cases the classes were specially tested owing to the known high percentage, e.g., (i) a backward class gave 15 per cent., whereas three other classes in the same school gave 5 per cent., 3 per cent. and 3·5 per cent.; (ii) Standard IA with 22 per cent., two other classes in the same school gave 3 per cent. and 6 per cent. respectively; (iii) Standard II with 13 per cent., whereas Standard I gave only 2 per cent.

From these results the question naturally arises: Why are there so many more left-handed children in mental defective schools? The answer to this question will be left for the present.

CHAPTER IV.—TWINS AND LEFT-HANDEDNESS.

In an elementary school two girls (twins) 14 years of age and nearly identical in all their characteristics—school work, appearance, &c.—were found on testing to be, one thoroughly left-handed, the other right-handed. On the next day in a mental defective school, twins—boy and girl—gave the same results, i.e., one left-handed, the other right-handed. Sometime later, it seemed probable that an investigation on the "handedness" of twins might throw some light on the hypothesis suggested later in this paper. There appear to be two kinds of twins—the one being derived by the fertilization of a single ovum, the other from the fertilization of two distinct ova. In the first case the twins are stated to have exactly the same characteristics, and it seemed that it would therefore be possible to decide whether left brainedness was one of these characteristics. As will be seen by the facts given below, it is by no means so simple a question as appears at first sight, as it is quite possible that the two twins may originally have been "identical," but that one may be very unlike the other in some of the characteristics (such as height, mental capacity, &c.) owing to some accidental cause.

Galton [8] in speaking of twins states that "the word twins is a vague expression, which covers two very dissimilar events—the one corresponding to the progeny of animals that usually bear more than one at birth, each of the progeny being derived from a separate ovum, while the other event is due to the development of two germinal spots in the same ovum. In the latter case they are enveloped in the same membrane, and all such twins are found invariably to be of the same sex."

The following results were obtained from 219 pairs of twins, most of whom were in ordinary elementary schools. They were all tested in the usual way described in this paper.

TWINS.

Sex	No.	Both Right-handed	Both Left-handed	One Right, other Left-handed
Boys ..	59	47	0	12
Girls ..	79	69	1	9
Boy and girl	81	53	3	25 (i.e., 11, boy R. and girl L.; 14, girl R. and boy L.)
Total ..	219	169	4	46

It is remarkable that in the mixed twins (boy and girl) nearly one-third the number consists of the one right-handed and the other left-handed. As to ability, appearance, &c., about 66 per cent. both of boys and of the girls are very much alike, when both are right-handed, and 33 per cent. are very dissimilar.

Some curious facts were noticed in the case of twins, one of whom was left-handed and the other right-handed, facts which are shown in the following table of comparison of the left-handed twin with the right-handed. The notes as to ability and physical defects were obtained from the teachers before the tests were made.

It will be noticed that in eight cases the left-handed twin is mentally defective and the right-handed is in an ordinary elementary school. Hitherto no case has been reported of the right-handed twin being in a mental defective school and the left-handed in an ordinary school. Not infrequently both right-handed twins are in a mental defective school. In twelve cases the left-handed twin is very nervous, much shorter, or more retarded compared with the right-handed.

It is possible with some of the twins (left and right-handed)—

(1) Especially where both are quite normal, that the left-handedness of one may be due to some accident as given in the note (p. 335), or

(2) Where the right-handed is much retarded, that they were originally both left-handed, e.g., in case of chorea, &c.

Comparison of Twins, one of whom is Left-handed, and the other Right-handed.

Both Boys.

	The left-handed twin	The right-handed twin
1	Very nervous	Normal
2	Very nervous, cries, shorter, less intelligent	,,
3	Normal	Rather retarded (a brother is left-handed)
4	Very short, in Standard IV	Very tall; in Standard IV
5	Left eyelid droops; in Standard IV ..	In Standard V
6	Healthy; average ability	Rather dull; adenoids and nasal catarrh
7	In mental defective school; defective speech	Died at 9 years of age; ordinary school
8	In mental defective school; hesitates in speech	In Standard IV
9	In mental defective school; 15 years old ..	Was in mental defective school; allowed to leave at 14 as much progress made

Both Girls.

1	Normal (very alike)	Normal
2	Dull	Dull; chorea
3	Much shorter and more babyish	Ordinary
4	In mental defective school; 8 inches shorter than sister, and much less developed	In mental defective school

Boy and Girl.

	Girl left-handed	Boy right-handed
1	Very slow; "peculiar speech"; improving, now writes right-handed	Normal
2	Chorea	,,
3	In a mental defective school	,,
4	Examined for mental defective school; on probation; very small	Normal, small
5	Below average intelligence	Dull
6	Very bright	Comparatively dull
7	Normal	Normal
8	Good ability; shorter, delicate; glandular abscess in neck and leg	Better ability than sister
9	Standard V	Standard VII; suffers from heart
10	Backward; lungs affected	Very bright

	Boy left-handed	Girl right-handed
1	In mental defective school	In mental defective school
2	,, ,, ,, ...	Below normal
3	,, ,, ,, ...	Fairly intelligent
4	Above normal	Above normal
5	Average ability	Below average ability
6	Not so bright as sister	The brighter
7	Normal; left eye squint	Normal
8	Standard VII	Standard V
9	In mental defective school; very defective speech	Standard V
10	In mental defective school	Standard VI; delicate
11	Much shorter than sister in infant school ..	Under-sized; in infant school

Note—In the statistics given are included all the twins in elementary and mental defective schools, who have been tested. Among the twins classified as both right handed were:—

(1) Both girls, in an infant school, one did most things left-handedly, but she was right-handed until she broke her arm three months previously.

(2) Both girls, aged 13½ years . one left-handed in everything except writing. She was right-handed until she burnt the palm of her right hand three years ago ; she has only just recovered, the palm is very badly scarred.

Chapter V.—Mirror Writing.

As has already been mentioned, many children in mental defective schools were found to write mirror fashion when writing with their left hand, and as this kind of writing was prevalent in these schools, various experiments and inquiries were made in reference to the matter. The ages of the children who wrote mirror fashion varied greatly—from about 10 to 16 years; younger children could not as a rule be tested, as they were not able to write their names. With one or two exceptions the mirror writers were right-hand writers, but it was remarkable that the left-hand mirror writing of most of these children was much better than their ordinary writing with the right hand.

As a matter of fact, the mirror writing in the case of 56 per cent. was very good indeed, of 29 per cent. good, and 7·5 per cent. fair, and of 7·5 per cent. bad.

57 per cent. started at the right-hand edge of the paper, 13 per cent. in the middle, and 30 per cent. towards, or not far from, the left margin—the usual place of beginning in ordinary writing. At first, there seemed to be a close connection between the act of mirror writing and the starting position; e.g., one began correctly, but after writing the first letter, which was at the extreme edge of the paper on the right, finished mirror fashion. Another began at the extreme right edge and wrote each letter correctly, but from right to left, i.e., backwards.

Some began mirror fashion, and apparently noticed that something was wrong, and then tried to correct themselves; in some cases successfully, but in others without success, even after trying two or three times. Their hand appeared to be doing exactly the opposite to what they wished. The majority were unable to read what they had written, but many could read an easy word, such as "cat" written mirror fashion. As a rule, the writing was done very rapidly and without hesitation, even by the very "low grade" feeble minded. But there were other cases where it was evident that the child was following carefully with the eye every letter, and it seemed certain that the letters were being formed in exact accordance with an optical mental image formed at the time. Generally the writing in these cases was slow, but it was well done. How remarkable this mirror writing is can be easily understood when it is explained that intelligent and capable teachers, who have not practised mirror writing, find great difficulty in writing in a "running hand" a capital (not printed) G or L mirror fashion, and yet these very mentally defective children could do it without any difficulty or effort.

A few print their names (mirror fashion), but as a rule the writing looked at in a mirror is very similar to the ordinary writing of the child. In one or two cases the name is printed with the right, and written in a "running hand" mirror-fashion with the left; these latter cases are in schools which have lately taken up "print writing."

One boy, a left-hand writer, wrote mirror fashion with his right hand. There were there cases of the same kind, and also mirror writing with the left hand by children who habitually write with the same hand.

Various experiments were made with the children who wrote mirror fashion, e.g., they were asked to write "cat," "10," "25," &c, with their left hand and then with their right. In most cases, the left-hand writing was mirror fashion, and the right in the ordinary and correct way. Sometimes the right-hand writing was done first, but the results were the same, namely, mirror writing with the left.

Again, others were asked to write "cat," "dog," "45," with the right hand, and then to copy them underneath with the left; the result was the same. The writing was done as a rule without any hesitation, and with rapidity. A word, e.g., "cash," was written mirror fashion, and some of these children were asked to copy it underneath with their right hand; they generally copied it correctly, i.e., not mirror fashion; on being asked to copy what they had just written, but to do it with the left hand, they wrote it mirror fashion. In one experiment the word "illustrate" was written mirror fashion in a running hand by the experimenter. The child could not read it, but when asked to copy it with her right hand she immediately wrote the word correctly (not mirror fashion) in every detail.

There are a few letters and figures which are frequently written mirror fashion by young children, especially s, d, b, 4, 6, and 9, but this writing is undoubtedly different from the mirror writing under discussion, and may be due to the lack of accurate observation on the part of a child, and never seems to extend beyond single letters and figures; but of course correct writing with one hand and mirror writing with the other is due to some other cause.

At one school, a little girl (D. E.) copied in print a whole page from a book mirror fashion, with the exception of "a" and "e"; she had never done this before, and she had not done it since. She was tested in the usual way with others in the class; she "rubbed," "threw," and "wound" with her left hand, but "picked up" with her right, and could only "cut" with her right; her "grip" was 2 kilos with each hand; she has a "peculiarity" in her speech. On thinking the matter over, it seemed probable that she had done the copy with her left hand, so at the next school, where two mirror writers were found in one class, they were both asked to copy out a page from a book, and to do it with the left hand. They were both right-hand writers, but they made the copy rapidly—one was well and correctly written, the other was well written, but every letter and word was written mirror fashion. This girl (E. A.) aged 14, is hydrocephalous and very defective; she "threw," "picked up," and "wound" with her left, could "cut" with either hand, her "grip" was 12 kilos with each hand,[1] but she always "rubbed" with her right hand. At a subsequent visit to the first school, the little girl (D. E.) was asked to copy some lines from a book with her left hand, and to begin at the right-hand corner. She immediately wrote her name mirror fashion, and then wrote a whole line in a similar way. When she reached the end of the line, i.e., at the left-hand margin of the paper, she asked whether she should go back the other way, that is, starting just under where she had finished. She then wrote this line quite correctly, not mirror fashion. The same experiment was tried with her right hand, only she was asked to begin in the middle of a line. She wrote correctly until she reached the end of the line, and was then asked to go on writing from the position just under where she had finished; without any hesitation she wrote this line mirror fashion with her right hand. This little girl was 9 years 2 months old. The doctor reported when she was examined (aged 8 years 8 months): "Talked at 5 years; walked at 2 years. Defective articulation: left-handed." Until three months ago she used to write with her left hand, but not mirror fashion. After a few months writing with her right hand, she writes mirror fashion, when she reverts to her left hand. Further, she now writes mirror fashion with her right hand when she is started at the right edge of the paper. It would therefore appear that in her case at all events the change in writing from one hand to the other may have had something to do with the peculiar kind of writing. As will be shown later (p. 342) a very high percentage of children, who have at one time written with the left hand but now write with the right, write mirror fashion when asked to write their names with the left hand. It is important to note that this girl did not write mirror fashion when she first began to write with her right hand, but that it was only after she had practised writing with her left and also with her right hand. The importance of this fact will be understood when the hypothesis suggested by Baldwin is considered (p. 353).

Subsequently it was found, especially in the case of children, who at one time had written mirror fashion, and of others who sometimes write with the left hand, and at other times with the right hand, that they can be made to write mirror fashion by merely asking them to begin at the extreme right margin of the paper. The probability of such a result only suggested itself quite lately, so only a few experiments have been made. Teachers were asked for the names of children who sometimes write with the left and sometimes with the right hand. The following results were obtained from a few children selected in this way.

---

[1] Normal grip for a girl of this age: right 24·79, left 22·92 kilos.

H. N., boy, aged 8. The doctor reports: "Speech defective. Father epileptic (?), and cannot read or write." The boy when asked to write his Christian name with his left hand beginning at the extreme right edge of the paper, wrote in mirror fashion without any hesitation. He wrote his name correctly with the same hand, when asked to begin at the left margin of the paper. With his right hand he did exactly the same, i.e., mirror fashion at the right edge, and correctly at the left. There was, however, one peculiarity in all the four written names, namely, the correct writing (not mirror fashion) of the final "y" in his name (Henry). At another school very similar results were obtained with a left and right hand writer. This boy with his left hand wrote his name completely mirror fashion without hesitation when started at the right margin, also "cat," but "25" correctly; with his right hand, however, under the same conditions, he hesitated and seemed to find some difficulty in beginning, but finally wrote his name completely mirror fashion. At the left margin, he wrote his name correctly with either hand. Other experiments were made with left-handed children, but some of them could not be induced to write mirror fashion with either hand, when asked to begin at the extreme right margin, as they insisted on edging off to the left and then wrote their names in very small letters, so as to get them into the only available space. Other mirror writers (with left hand), many of whom wrote mirror fashion wherever they were started, could not be induced to write mirror fashion with the right hand under any circumstances.

Another interesting case was that of a boy (H. W.), left-handed in writing and in nearly everything else, except throwing, who wrote all letters and numbers correctly with his left hand, but wrote them mirror fashion with his right.

A curious case was met with at another school. A little girl (L. C.) wrote a word correctly with her left and mirror fashion with her right: she was asked to write another word first with her right—she wrote it correctly; but she wrote it immediately afterwards mirror fashion with her left hand. She is 12 years 9 months old: throws left, cuts left only, winds left and writes left. The doctor reports: "Infantile paralysis, right hemiplegia, used to write mirror fashion with her left hand; gave it up in 1916 (i.e., about 8 years of age), but reverts to it at times in 1917." At a second visit she printed her name completely mirror fashion on the blackboard with her right hand when asked to begin at the right-hand corner.

Early in the investigation it was noticed that there was a much higher percentage of mirror writers among children, who, although they wrote with the right hand, were evidently left-handed in many others of their activities. This fact has been noticed by previous investigators. Ballard (1916), referring to mirror writing, says: "With rare exceptions it is done by left-handed people only, and with the left hand only. . . . The subject does not at first recognize the word as he writes it down; nor can he without some practice read it. . . ." [4].

A. L. Beeley (Instructor in Psychology, Utah University), after an investigation in schools of Chicago in 1918, came to a similar conclusion, namely, that mirror writing is "a characteristic of extreme left handedness," and "not necessarily of mental deficiency." Ireland [11], 1881, connected mirror writing with left-handedness.

As mirror writing appeared to be associated with left-handedness, many left-handed adults were asked to write their names with the left hand, but they always did it correctly. In one case, however, it was noticed that the pencil seemed instinctively to have been placed at the right-hand edge of the paper, and then withdrawn and the writing begun at the usual place—left margin—without the "subject" paying much attention to what she had done. An experiment was next made with a left-handed teacher, a capable and intelligent head-mistress. She was asked to write her name with her eyes shut; she did it rapidly with her right hand, and quite as well as with her eyes open. She was then asked to close her eyes, and write her name with her left hand; she did so immediately but mirror fashion. When she looked at it, she could not make out what she had written, and said, "What is the matter with me?" When the paper was held up to the light—the reverse side—an excellent copy of her name in her ordinary writing was seen. She explained that she was quite sure she was writing it in the ordinary way.

Similar experiments with a few other left-handed teachers were made, and in one case with exactly the same result. In fact the latter, a capable and intelligent assistant teacher, made nearly the same remarks as the first. In still another case, although the teacher wrote her name correctly with her left hand, she stated that while writing it she thought at times she was writing it "backwards." A few left-handed adults who write with the right hand were found to be able to write mirror fashion without any difficulty with the left hand, although they had not made the attempt before. Others—right-handed or left-handed—have to concentrate their whole attention on what they are attempting, and even then make mistakes.

McHattie (1913) [20] describes some experiments he made in London elementary schools in connection with mirror writing. He found in the left-hand writing of 1,240 children, ages 7 to 14 years, that there were only seven cases (0·48 per cent.) of mirror writing. One was a girl, she was not included in the percentage, whose right hand had been partially disabled by infantile paralysis, and could not write with this hand. The percentage was about twice as high with the boys as with the girls.

In the present investigation 829 children were tested in left-hand writing in the ordinary elementary schools (infant and senior departments), and only four (0·48 per cent.) were found to write mirror fashion. The total numbers in both cases are small, but they agree very closely, and it appears likely that the percentage (0·4 to 0·5) is not far wrong. A. L. Beeley [6], in elementary schools in Chicago in 1918, found in 103 schools with a school population of 106,356, forty-two mirror writers—i.e., about 0·04 per cent. In his investigation, however, he did not test the left-hand writing of every child, but only asked for information from the teachers concerning all children who had been observed to write mirror fashion at any time in their lives. There was about the same percentage of mirror writers among the boys as among the girls.

The percentage of mirror writers in mental defective schools in London and Middlesex is given below:—

Number of children treated	Number of mirror writers	Percentage
1,350	109	8·0

These numbers include both boys and girls of varying ages from 8 to 16 years, generally about 8 years of age and upwards, as only those who could write their names were tested. Mirror writing of isolated letters and figures—such as 3, b, d, 4, 6, 9—is not included in the above percentage. It is evident, therefore, that mirror writing is very much more prevalent among the feeble-minded than among normal children; the percentage is in fact about seventeen times as great.[1]

A still more striking result is obtained from the calculation of the percentage of mirror writers among left-handed children.

The following statistics have been carefully made out in the case of mirror writers, who had all been tested in "handedness."

---

[1] In one school thirty-one were tested, and there were ten mirror writers—a most remarkable result. It seemed possible that the mirror writing might have been due to some special methods of instruction in writing, drawing, physical exercises, &c., but after careful investigation it was evident that the methods used were the same as those used in other special schools.

Mirror Writers among Left-handed Children.

Sex	Number tested in left-handed writing	Number found to be left-handed	Number of mirror writers among left-handed	Percentage of mirror writers among left-handed
Boys ..	374	61	11	18
Girls ..	355	76	30	40
Boys and girls	729	137	41	30[1]

That is to say, 30 per cent. of the left-handed children were mirror writers, and in the case of girls the percentage reached the astonishing number of 40 per cent.

Further the mirror writers were nearly always left-handed children, who wrote with the right hand. These percentages were further analysed with the following results: 40 per cent.[2] of these left-handed boys habitually write with the left hand, and 60 per cent.[3] with the right hand.

Of these left-handed boys who write with the right hand 32 per cent. wrote mirror fashion with the left.

Curiously enough the same percentage (i.e., 40) of girls write habitually with the left and 60 per cent. with the right hand.

Of these left-handed girls who write with the right hand, 62 per cent. wrote mirror fashion with the left. The majority of mirror writers have, therefore, at one time or another written with the left hand; and it would seem that, this being the case, they would be less likely to write mirror fashion than children who had never co-ordinated their left-hand movements to the usual forms of letters and words.

It is therefore evident that mirror writing is very closely associated with mental deficiency,[1] and also with such left-handedness as has been brought to light by the tests already described. It is also clear that mirror writing is not itself due to mental deficiency alone, as only 8 per cent. of the feeble-minded write mirror fashion, nor is it due to extreme left-handedness alone, as very few left-handed children in ordinary and in physical defective schools write mirror fashion. Further, it does not seem impossible that the same cause may in some cases have brought about left-handedness and also mirror writing; that in fact something may have affected the dominant (in these cases the left) hemisphere. It may have been a lesion, causing among other things slight hemiplegia, or it may have been due to defective development of the dominant hemisphere. It would then follow that such left-handedness differed from what is usually termed "natural" left-handedness.

---

[1] The percentage of right-handed children (i.e., children who gave little or no indication of left-handedness in the tests) who wrote mirror fashion with the left hand, was of boys 1·6 per cent. and of girls 2·8 per cent.

[2] To see whether this result gives a fair estimate, a larger number was taken: of 251 found left-handed 106 habitually write with left = 42 per cent.; 51 are known to have written left = 20 per cent.; 94 are not known to have written left = 38 per cent.

[3] There is a peculiar coincidence in connection with the children in mental defective schools who habitually write with the left hand. As has already been stated (p. 332) 18·2 per cent. of the children in mental defective schools were found by the various tests to be left-handed and, as it is estimated above, 40 per cent. of these are left-hand writers: it therefore follows that 7·2 per cent. of the children in mental defective schools write with their left hand—a number almost identical with percentage of left-handed children in ordinary elementary schools—namely, 7·3 per cent. (see p. 332). It seems, therefore, not unlikely that many of these (7 2 per cent.) may be the "naturally" left-handed children—children whom it has not been found possible to change into right-hand writers.

It is very necessary, therefore, at this stage to consider more carefully what is meant by "left-handedness," and also its various forms and their causes.

There has been much discussion and controversy, since Broca's investigations on aphasia, as to whether there is a dominant side of the brain, and whether its dominance is indicated by left or right-handedness. The origin of the controversy arose from observations of a large number of cases of aphasia associated with right hemiplegia, especially paralysis of the right hand, and of a much fewer number with left hemiplegia. Whether the motor speech centre is located in the third frontal convolution, as suggested by Broca, or in the lenticular zone as contended by Marie, or elsewhere, it is unnecessary to discuss in this paper. But it is agreed upon by all that in the majority of cases it is located in the left hemisphere, and further it appears to be held by most authorities at the present day that not only is the speech centre, but that also other important language centres are located in the dominant hemisphere: for the right-handed in the left hemisphere, and for the left-handed in the right.

Moutier in 1906, however, contended that the percentage of cases of left hemiplegia associated with aphasia was much less than ought to have been the case, if this theory was true; and in addition he cites six cases where the aphasia was associated with hemiplegia of a kind opposite to the known handedness of the patient. Moutier's estimates, however, of the percentage (10-12 per cent.) of left-handed are much higher than those usually recorded. In the cases he has collected (1864-1907) and given in his book, the percentage of cases of aphasia associated with left hemiplegia appears to be about 8·0 per cent. (total cases 347, 318 aphasia with right hemiplegia and 29 with left hemiplegia). He also quotes the following results as given by Hammond (1871): 17 cases of aphasia with left hemiplegia (i.e., 6·4 per cent.) and 247 cases of aphasia with right hemiplegia. In respect also of eighty-two autopsies, in two cases (i.e., 2·4 per cent.) the lesion was located in the right hemisphere. Taking the above 676 cases in all noted, of these there were forty-eight in which the right hemisphere appeared to have been affected. This gives a percentage of 7·1 per cent., a number almost identical with 7·3 per cent., the percentage of left-handed children found in elementary schools during the present investigation. This latter percentage (7·3), as will be seen later, is probably too high, when left-handedness is considered from the point of view of a dominant hemisphere. It must be again emphasized that the above number (676) is far too small a one on which to base a percentage in a matter in which there is such a great variability, as has been shown to be the case in left-handedness.

---

[1] A. L. Beeley [6], as already mentioned, considers that mirror writing is not necessarily a characteristic of mental deficiency. This is no doubt true, but it is certainly more prevalent among feeble-minded than among normal children.

Again, in reference to question of dominance of one hemisphere, Laurence (1869) asks the pertinent question: Why does atheroma (degeneracy of the walls of the arteries) more often attack the vessels of the left hemisphere?

If lesions among adults—apart from tumours, injury, &c.—are found more frequently in the left hemisphere, and in a proportion even approximating to the percentage of the right-handed, then such a fact would seem to be a strong argument in support of the theory of a dominant hemisphere, and of the belief that the dominant side is the one that is more likely to give way under strain in certain diseases of the brain; that in fact the hemisphere that experiences the greatest strain will be the one to break down first.

If there is a dominant hemisphere, and if this dominance is revealed by "handedness," then those with a dominant left hemisphere will be right-handed, and those with a dominant right hemisphere will be left-handed. There will, however, be some who have been driven to the use of the non-dominant side in all or only in some of their activities by various causes, such as are given below.

Various Causes of Changes of Handedness.

(i) *Very simple causes and requiring no explanation.*—(*a*) Loss of right arm, hand, &c., even the loss of a right thumb, appears to have been sufficient to make a child completely left-handed, e.g., a child without right thumb gave the following results, when tested: rubs, throws und writes with left hand, and of course can only use her left hand in cutting with the scissors. Again, a head teacher stated that she is very left-handed, but writes with her right hand; and when tested she gave every evidence of left-handedness. Subsequently she was told by her mother that she was not originally left-handed, but that at 3 years of age she had very seriously injured her right thumb-nail, an injury which prevented her using her right hand for nearly three years. (*b*) Broken right arm: girl, age about 7 years, broke her arm a year ago, attended three months with her arm in a sling. Tested: could cut with left and right, and could write equally well with either hand; in everything else was right-handed. (*c*) Injured right hand: girl, Standard I: writes with left hand, as she had once injured her right hand.

(ii) *Injuries to the nervous or muscular systems.*—Various forms of hemiplegias; in some cases the affected hand cannot be used at all, in others to a limited extent, and in still others the effect is apparently only transitory, and perhaps often unnoticed. All degrees seem to be found in mental and especially in physical defective schools. It would seem that in some cases the affected hand has continued to be used in writing throughout, but that the unaffected hand has been resorted to in many other activities. A very interesting case was met with in one of the mental defective schools, the history of which is given as fully as it could be determined. Like most of the cases met in these schools it is very difficult to obtain accurate information, especially from parents, with reference to dates.

H. H., girl, born April 13, 1906 (even this date was uncertain; it was given at one school as April 13, 1905, and might have been April 13, 1907). Walked at 1 year 10 months, talked at 1½ years; entered an infant school in 1910 about 4 years of age. The head teacher remembered the girl as being normal and bright, and rather "chubby," and not left-handed. From there she went to the seaside, and then reappeared at the girls' school (senior department of the infants' school) on September 22, 1914, i.e., at 8½ years of age, and stayed until October 1, 1914. The head teacher remembered her as not being at all mentally defective, but troublesome and a bad influence on the younger children; and for this reason she was placed among older girls, Standard V(a); she was in this school only eight days. She next entered another girls' school in the neighbourhood on November 9, 1914, and remained there until November, 1915. She was placed in Standard I. The attendance registers showed that she had made very regular attendances until May 21, 1915. After that date no attendances are recorded until September 21, 1915, and after that the attendances were very irregular until November, 1915. On May 21, 1915, she was apparently 9 years old.

Her mother states that the girl fell from a chair on her head, when she was 8 years old (9 years?) and was insensible from 6 p.m. to 10 p.m.; that this fall produced concussion of the brain: that "there was a bruise on the left temple," and that "the left leg has since had a lot of veins outstanding"; that "she was silly for about four months suffering from loss of memory." It would, therefore, seem probable that the accident occurred on May 21, 1915, as everything points to this date except the assumed age. The head teacher remembered her as being not very bright (as she remained in Standard I for a year), and she thought there had been some accident. The teachers did not think she was left-handed. In December, 1915, i.e., at 9 years and 8 months, she was examined by the medical officer and sent to the mental defective school on January 11, 1916.

At the original medical examination the doctor reports: "Father and grandfather suffer from rheumatism. Child, giddy and hysterical; fits not epileptic (major); an accident nine months ago: concussion of brain by fall, unconscious four hours. Reading, only little words; writing, scarcely legible; drawing, very little." The head teacher of the mental defective school reports, that the girl often makes grimaces and complains of headaches, and her hands are unsteady, and that the mother now states that there is no family history of fits, and that the girl "uses both her hands; she used to use the left hand more," but that the mother does not remember when she first noticed the use of the left hand. Probably if the child had been born left-handed the mother would have remembered the fact. Everything seems to point to the fact that the girl was, until the accident, right-handed. The real evidence of this is the fact that she could hardly write legibly, when she was examined by the doctor at 9½ years of age; such a fact would undoubtedly have been noticed at her former schools. When tested in December, 1919, the following results were recorded: Aged 13 years 8 months, rubs left, throws left, cuts left and right, picks up left, winds left; dynam. left = 38, right = 44; writes right but wants to write left.

She wrote her name mirror fashion with her left hand. Unfortunately she was of course never tested in these things before the accident, but it is extremely unlikely that she was then either left-handed, or wrote mirror fashion (as only 4 or 5 in 1,000 do so). She now writes very well with her right hand, and it will be interesting to discover whether she changes in any of the tests as she grows older. The average grip for normal girls is as follows: for 14 years of age, right 24·79; left 22·92.[1] Her right and left hand grips are therefore far above normal.

---

[1] Smedley's investigations [28].

S. S. writes with the right hand, but "used to wish to write with the left, owing to paralysis of right hand; the paralysis has much improved." When tested, he rubbed, threw, and picked up with the left; he cut easily with his left, but not very well with his right hand. This, therefore, is the case of a boy who was slightly paralysed, but not so badly as to prevent him writing with his right hand.

A. H. G., a boy, aged 6 years. Had a very serious accident when 9 months old from burning, and was not out of danger for over three months; was noticed by mother and nurse to be very left-handed before and after the accident. An intelligent child: at 4½ years of age began to stammer badly with twitching of eyes; both continued until 5¼, when the stammer gradually got less and finally disappeared. At 6 years of age he gave hardly any evidences of left-handedness, except that of holding a glass in his left hand while drinking: he always held a bat in his left hand when knocking about a ball. At 7 years old he appeared to be thoroughly right-handed except in one or two small things. Instruments were used at his birth. It is suggested that some cause affected temporarily the hand centre, &c., and that the passing away of this allowed him to revert to his natural handedness.[1]

One or two further references in connection with this question of dominant hemisphere may be of interest.

James Kerr, 1920 [16], writes: "The right hand has generally a greater capacity for fine adjustments and co-ordinations than the left, which last is then accessory. There is at present no worthy explanation of why the right hand should be so specially used, except that it is driven directly from the left side of the brain, and that in the majority of all races that side gives evidence of a higher state of functional evolution than the other." "The interpretation of sights and sounds into ideas and the intellectual functions which control the voluntary machinery of the organism are commonly bound up with functioning of a highly developed left cerebral hemisphere, so that in normal individuals the left hand, although its motor centres are on the right side, is still driven indirectly through these right-sided cortical centres by the higher control of the left side." And again: "The specialized aptitude or capacity associated with psychical phenomena of speech and reasoning which is so marked in the left brain is best described as dextrality. In a small proportion of individuals the reverse happens: instead of the usual high functional development of the psychic qualities in the left brain it is the right side which shows this property; a consequence appears that the nearest motor centres, those of the left hand, become the most specially used and the right remains accessory."

---

[1] "In these cases of congenital hemiplegia and decided left-handedness (which I believe is not infrequently the result of injury to the left cortex). . . ."—G. Vivian Poore [1].

Josefa Ioteyko (1917) [10] asserts that it has been proved that the asymmetry extends not only to the centre of projection, but to centres of language, recognition of objects and signs, purposive activity (the loss of which results in ataxia), and that the localization of these centres is in the left hemisphere for right-handed people.

Further it appears to be true that there is an increase in the percentage of left-handed, beginning with normal children in elementary schools (7 per cent.), increasing in the case of feeble-minded to 18 per cent. and rising to 16 per cent. to 30 per cent. in the case of idiots and epileptics.[1]

In connection with the above statements some very curious facts were noted during the present inquiry, facts which at the time appeared to be very contradictory. Very frequently the teachers in ordinary schools asserted that the children found to be left-handed were the most intelligent and the best at school work; in fact some were stated to be the best in the school. Equally frequently the teachers in the mental defective schools described the left-handed to be the dullest, and of the lowest grade. Ballard, who has special knowledge of left-handed children, states in 1916 that left-handed children compare favourably with right handed in intelligence and school attainments, but he adds that left to right "transfers" (i.e., left-handed children made to write with the right hand) are inferior, and have a tendency to stammer.

One very curious remark was made by the head teacher of one of these special schools, and a special note was made of it at the time, as it appeared to be opposed to what was then thought to be likely, namely, that the change of left-handedness to right-handedness was accompanied by harmful results. The head teacher stated that she had frequently noticed a great improvement in a child's intelligence and school work associated with a natural change of left to right-handedness. There is a very high percentage of left-handed in this school, and no efforts are made to bring about a change. A similar change has been noticed in other schools, an example of which is given below[2]:—

E. Y. (aged 8 years 2 months) throws left, cuts left and right, picks up left, winds left. Copied her name mirror fashion with left, as cannot write it spontaneously. Doctor reports: "Talk four years; walk three years; left-handed at age 7 to 8 years." Head teacher reports: "She is changing from left to right hand in writing without any suggestion from the teacher, and she has begun to improve generally."

---

[1] Mayet in 1902.

[2] Ireland, W. W. (medical superintendent of Scotland National Institution for Imbecile Children, Larbert), notices a similar change of "handedness" among imbeciles [12].

There seems therefore to be good reason to believe that there are certainly two distinct types of left-handedness:—

(1) Those who are naturally left-handed, i.e., those who have a dominant right hemisphere.

(2) Those who are naturally right-handed, but who have been driven to the use of left hand in some of their activities owing to some defect of the nervous or muscular systems.

In the case of certain children in the mental defective schools, probably something more than the hand centre or its nerve fibres, &c., has been involved. It also seems probable that the percentage of naturally left-handed in mental defective schools may be no higher than that in ordinary schools, the increased percentage being due to some cause that has brought about the change, before birth, at birth, or at some subsequent date.

These conclusions, as will be remembered, had their origin in the discovery that mirror writing was very prevalent among left-handed children in mental defective schools. A further discussion on mirror writing may therefore be of value. The following are some of the conclusions of other investigators on the subject:—

"In 200 apparently healthy people, only the neuropathic wrote mirror fashion; in 200 sick subjects only those suffering from nervous disorders wrote mirror fashion" [15]. Soltman found (number tested 77) 35 per cent. were mirror writers among deaf-mutes.

Kerr [16] states: "Mirror writing also occurs pathologically with the right hand, being common among hemiplegics and others with brain disease, although even here unusual with better class or well educated individuals. It is to be observed frequently in the developing child. Children becoming deaf soon after birth are said to mirror generally with the left hand, but if the deafness supervenes later the mirror writing does not occur."

McHattie quotes that mirror writing is frequently observed in nervous diseases, e.g., hemiplegia, hysteria, and locomotor ataxy. Among his own observations he gives the case of a girl whose right arm had been partially disabled by infantile paralysis, and who could not in consequence write with her right hand [20].

J. K. Fuller [7] (1916) gives four cases of subjects under hypnotism, three of whom wrote mirror fashion with left hand; one of these is definitely stated to have no inclination to left-handedness.

C. H. Judd also states that "Certain persons when hypnotized produce mirror writing with the right hand" [21].

F. T. Allen (1896) [2] says that mirror writing was discovered when nervous disease rendered normal writing difficult or impossible, and he concludes that it is often a symptom of nervous disease.

F. B. Sherlock [24] writes: "One finds rather widely prevalent the notion that a characteristic of idiocy is that whereas the normal person writes from left to right and with the right hand, the idiot writes from right to left with the left hand . . . the script is meaningless until it is held before a mirror.

"Good examples of this kind of writing are rare, at any rate in this country (England); for idiots do not write at all, or they produce a scrawl, which demands the use of a good deal of ingenuity, in addition to a mirror, if anything is to be made of it.

"Ambidexterity, as we shall see, is commoner in idiots than in normal persons, and the particular accomplishment which we are considering seems to be a special case of it."

Although the following quotation (Kerr [16]) does not specify mirror writing, it seems to indicate that such peculiarities may be expected in such a class.

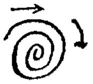
"Whilst in the last class (i.e., defective functioning of both hands) are individuals whose dextrality has not been properly developed, and who are in this sense mentally defective, and on further examination may be expected to present not merely gaucherie, but speech defects, and the various degenerative stigmata in relatively large proportions."

Various explanations have been given as to the cause of mirror writing, but none of them are altogether satisfactory, and they do not account for many of the known facts.

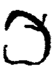
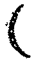
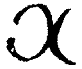
There is no doubt a tendency for the left hand to move in a way similar to movements made by the right hand, e.g., in the simple case of drawing two circles at the same time—one with the right, the other with the left hand, as is illustrated below:—

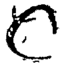
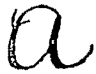
Left. Right

Some experiments were made in the ordinary elementary schools on the effect on the left hand of right-hand practice. Young children 2 to 3 years of age when they begin to use pencil or chalk usually draw a series of circles, thus :—



The direction with the right hand is as indicated by the arrows, i.e. clockwise. If the pencil is held in the left hand the direction is exactly the opposite, i.e., anti-clockwise. The first fact is well known in infant schools, as teachers have some difficulty in persuading children to write an "a" in the usual way, since nearly all begin by writing it as follows :—

 then adding  which gives 

instead of  and  as is usual among older children.

The following results were obtained by asking each child to copy a circle, drawn on a piece of paper or millboard placed before them, first with the right and then with the left hand. Care was taken that the children should not see what had been done by the others, and should not know what they were to draw until they were asked to do it.

Circle drawing with the left and right hand. Note, "N" indicates the natural way, i.e., clockwise with the right, anti-clockwise with left. "A" indicates the artificial way, i.e., anti-clockwise with the right, clockwise with left.

The results obtained with the right hand will be given first.

CIRCLE DRAWING WITH THE RIGHT HAND.

		Percentage of drawing in artificial way
Infant school, average age 4 years 4 months ..	33 children ..	27·2 per cent.
,, ,, ,, 4 to 5 years .. about	40 ,, ..	15·0 ,,

These had not been taught to write.

Infant school, average age 6 years 5 months ..	37 children ..	81·0 per cent.
,, ,, ,, 7 ,, 5 ,, ..	55 ,, ..	92·7 ,,
Boys' school, Class I, average age 12 years 5 months.	40 ,, ..	100·0 ,,

That is to say, these children showed a gradual change from the natural to the artificial way of drawing a circle on paper or millboard.

This change was undoubtedly brought about by the continued practice, in writing, of such letters as a, o, d, g, &c. The drawing of circles on the blackboard gives however different results, no doubt in part because it is an arm movement. In the above experiments, and in those that follow, the results given in the case of the babies (i.e., under 5 years of age) are very variable, and not very trustworthy. It would have been much better to have watched these children, and to have noted from time to time how in their ordinary drawing they formed a circle, as it often seemed a mere chance which way they drew the circle; sometimes due to the position of the hand, at others to that of the millboard. The same kind of thing was even more noticeable with their left-hand drawing.

Drawing Circles with Left Hand.

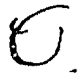
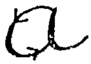
			Percentage who drew circle in artificial way
Infant school, average age	4 years 4 months	33 children	36·3 per cent. (not very reliable)
,, ,, ,,	6 ,, 5 ,,	37 ,,	21·6 per cent.
,, ,, ,,	7 ,, 5 ,,	55 ,,	21·8 ,,
Boys' ,, ,,	12 ,, 5 ,,	40 ,,	40·0 ,,

More often than not those who drew in the artificial way with the right still drew in the natural way with the left.

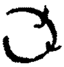
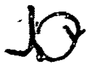
From these insufficient data however it would appear that in some cases the artifical way of drawing with the right had affected the left-hand drawing, as the children (classes ages 6 years 5 months and 7 years 5 months) who drew in the artificial way with the left were those who had also drawn in the artificial way with the right. In these two classes (92 children) only one—a boy—drew in the artificial way with the left, after having drawn the circle in the natural way with his right hand.

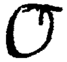
The elder boys show a greater change, as the artificial drawing with the left hand has increased to 40 per cent. Whether such a result would be confirmed by further experiments remains to be seen.

There is evidently a tendency, especially when both hands are used at once, for one hand to follow the other mirror fashion. This fact, however, in no way explains mirror writing of children when they begin to write, many examples of which are well known. As a matter of fact an "a" written by the left hand in the ordinary writing is in accordance with the natural tendency of that hand.

Thus  and completed 

To write this letter mirror fashion with the left hand would involve the forming of the circle in the artificial way with that hand, thus:—

 and completing thus: 

As a matter of fact, a fair number of mirror writers did actually write the letters "a" and "o" mirror fashion with the left, but the circle part was drawn in the natural way, i.e.,  .

A widely accepted explanation of mirror writing is given below, but it certainly does not explain all the facts; nor does it explain why so many more so-called left-handed children write this way with their left hand, unless it is suggested that left-handed children are more motor than visual inclined.

"If a man is of the so-called 'visual' type, i.e., if he depends mainly on his *v* series in his writing, the *look* of the letters, &c., and by comparing it with the resulting writing, conforming his movement series to it, then any movements which violate the figure presented by visual memory are unintelligible. Such a man must reproduce, with his left hand, the visual images as produced by the right. That is, he must write from left to right, with both hands, which involves symmetrical movements. This represents the power of the *v* series to bring the movements of both hands in conformity to it. If, on the contrary, his *m* (motor) series has grown independent by practice, and he remembers written words not by the way they look mainly, but by the way it feels to write them—if he is the so-called motor type in his hand writing—then his left-hand writing must produce the series of muscular sensations as his right hand has established. This represents the power of movements established by one hand to carry the other hand also with it in a symmetrical way. His left-hand position must duplicate at each moment his right-hand position, when he comes to try the experiment of writing in the air with both hands. This gives symmetrical movements with the two hands, which means mirror writing with the left hand"[1] [2].

This symmetrical movement of the two hands has been held by Fechner and others to be a strong proof that the discharge of energy into one side of the body tends to stimulate the corresponding members of the other side to similar movements. Lochte [18] and Weber [27] suggest that writing is primarily a motor act controlled by the optical image, which is a later development. Consequently if the mind and the eye are centred on the right hand, we should expect the left hand to follow out the movements of the right hand, and therefore write mirror fashion.

---

[1] J. M. Baldwin: "Mental Development in the Child and in the Race" (1895). A somewhat similar explanation was given by Goldscheider in 1892 [9].

E. J. Allen [2] remarks that the sensations accompanying all kinds of writing, whether right-handed or left-handed, are so similar as to suggest that they all start from the same region of the brain, but that there are a series of commutators at a lower level whereby impulses are turned into different channels. He further suggests that this supports the view that the true graphic centre coincides with either motor centre, but is superior to all.

Josefa Ioteyko (1919) [10] concludes from experiments made in her laboratory by Mlle. V. Kipiani, " . . . in the first place, that the movements acquired by the left hemisphere (which directs the movements of the right hand) are transmitted to the right hemisphere, and, secondly, that this transmission is not purely and simply an increase of excitability; but that it comprises all the complicated movements necessitated by the act of writing—in a word, that the characteristics of hand-writing certainly have their origin in some central cerebral cause."

Before considering further the hypothesis as suggested by Baldwin (p. 353) some examples will be given of mirror writing associated with various forms of paralysis, and with other physical defects.

M. S., aged 13 years 10 months, girl · "Mother in an asylum; in good health until 6 years of age, then a fit with paralysis, right hemiplegia. Reading nil; easy numbers, mentally only, can write fairly well with her left hand; no memory, speech slow and drawling (September 13, 1916); a few letters and words (September 6, 8, June 17). Sleepy, dull and exceedingly slow even for a mental defective; cannot spell "cat" (June 17). "Talks well about her own affairs." The doctor in his last report dated December 19, 1919, states that she is "word blind."

In all the tests that could be tried, she was of course thoroughly left-handed; she could cut easily with her left hand, her strength of grip was 30 kg.

At the first visit all the children in her class were asked to write their names with their left hand and then with their right. Her paper when given up had her name well written at the left side; but there was near the right margin a curious scribble, and emerging from it her name written mirror fashion; the writing was rather shaky. It was originally believed to have been written with her right hand. On a subsequent visit she was watched while she wrote, she immediately wrote her name with her left hand, beginning in the usual place; the pencil was then placed in her right hand, she had difficulty in holding and using it, but tried her utmost. After scribbling with an arm movement, she took the pencil in her left hand and wrote her name mirror fashion, rapidly and in excellent fashion. She was not known at the school to have written mirror fashion before.

L. C., girl, born April 20, 1907: infantile paralysis, very small for her age. Doctor reports paralysis of right leg and arm; writes mirror fashion with left hand; had given up mirror writing in April, 1916 (i.e., 9 years old); in 1917 (i.e., 10 years old) writes mirror fashion sometimes. January 19, 1920, when asked to try to write her name on the blackboard with her right hand, printed it very well, but all mirror fashion.

Mirror Writers in Physical Defective Schools.

M. A., girl, paralysed both legs. Mentally defective; tested: rubs and throws left; cuts left and right; wrote mirror fashion with her left hand.

J. S., aged 13 years 8 months, boy. Infantile paralysis right lower limb. Nervous. Cuts left and right; writes right. Doctor reports: "Leg flail-like though not very wasted." This boy tried twice not to write mirror fashion with left, but finally wrote both names completely mirror fashion.

A. S., boy, aged 11 years 3 months. Rubs and throws left, cuts left only; writes right, but sometimes left; is thought to be verging on feeble-mindedness. Doctor reports: "Right hemiplegia; slight weakness of right arm and leg, not wasting. Used to fall about a good deal."

J. S., boy, aged 14 years 1 month. Apparently right-handed, mirror writing very shaky indeed. Doctor reports, at 7 years 3 months of age: "Spastic paraplegia (partial); spinal paralysis; right ptosis; very intelligent; at 10 years 2 months, paralysis of eye and legs, getting worse; scissors legs." At present time school staff reports: "Not very intelligent."

E. G., boy, left-hand writer. Writes mirror fashion sometimes also 21 for 12, i.e., beginning with 1 and then writing 2 on the left of it; hydrocephalous.

C. R., boy, aged 8 years 3 months. Writes mirror fashion with left hand when tired. Doctor reports at 5 years 11 months: "Spina bifida, lower cervical region; night terrors; at 8 years 2 months, very backward: spina bifida apparently closed."

J. P., boy, aged 15 years 2 months. Throws left but writes right only; dynamometer left = 34, right 26. Doctor reports at age of 6 years 5 months: "(Brother dead, suffered from fits). Spinal caries (lumbar region), can pick up things. At 11 years 5 months pains in right leg. At 7 years 11 months no active disease; much lordosis, apparently habit."

F. B., boy, aged 8 years 4 months; very large head. Doctor reports "Infantile paralysis at 2 years 11 months; paralysis of both legs; right arm and leg not bad; operation for talipes. December, 1916, marked wasting of leg and thigh muscles. October, 1919, operation for tubercular disease in third metatarsal bone, sinus still discharging. Bright child."

F. S., boy, aged 10 years 11 months. Right-handed in all tests. Dynamometer, left = 33, right = 32. Doctor reports: "Spinal caries, lower dorsal, at 4½ years."

These nine, together with one left-handed girl suffering from mitral stenosis, who had been in a mental defective school for three years, were the only mirror writers among 706 children tested. It will be noticed that three of these ten were merging on mental deficiency, leaving seven among the physical defective children, i.e., 1·1 per cent. It is remarkable that in six cases there is some spinal lesion, or paralysis of the lower limbs; the seventh is that of a hydrocephalous child.

As reference will be made later to mirror writing by children; when they first began to write, the following cases are given:—

Examples of Children whose First Attempts at Writing were with the Left Hand and were reported to have been Mirror Fashion.

	Age	Sex	Rub	Throw	Cut	Writes	Doctor's report
	Yrs. mths						
W. S...	..	Boy	R.	L.	O.	L.	
H. S. ..	10 6	Boy	L.	L.	L.	L. and R.	"Bossed forehead; stunted; speech indistinct"
H. W.	8 8	Boy	L.	L.	L.R.	L.	"Speech inarticulate; decided epicanthus; mental age IV. (Binet)"
C. B. ..	10 8	Girl	L.	L.	L.	L.	"Defect of articulation; low bulging forehead"

Similar cases reported by other investigators.

A. L. Beeley [6], 1918, reports and gives details of three boys and two girls, all of whom wrote mirror fashion when they first attempted writing. They were all extremely left-handed and had, with one exception, left-handed near relatives. One, a boy, had not overcome the habit at the age of 7 years 11 months, as "he still frequently lapses into mirror writing."

If there is any truth in the suggestion that those who write mirror fashion with their left hand in mental defective schools are really right-handed (with a dominant left hemisphere), but have some defect in this hemisphere or in the nerve fibres connecting it with the opposite side of the body, a defect which has temporarily or permanently affected the children, then the few who, although they wrote mirror fashion with their left hand, yet in nearly every test reacted right-handedly, would probably display some left-handed activities in some of eight years, should suddenly write mirror fashion with the same hand, after making an effort with her right hand.

Also other cases of mirror writing by left-hand writers who had suffered from right hemiplegia from an early age.

(5) Why "certain persons when hypnotized produce mirror writing with the right hand." Should this statement be true, only one explanation seems possible, if it is to be in accordance with the above hypothesis, namely, that these "subjects" were really left-hand writers.

There also seems no reason to believe that left-handed children (even among the feeble-minded) should be more frequently of the "motor" than the so-called "visual" type. There is however this to be said, that a child with a fairly skilful left hand may be able to write with this hand without paying so much attention to what is actually being written, and for this reason might be inclined to trust to a feeling of the movements of the hand rather than to the actual forms of the letters and words written. On the other hand it has been pointed out that some children have actually attempted to correct their movements, thus clearly indicating that they had noticed that there was something wrong. These attempts, however, were not always successful. One or two examples of such attempts are given below.

(1) A boy wrote the first letter of his name mirror fashion; he then started again and wrote the first two letters of his name in the same way. Even after these two unsuccessful attempts he finished by writing both his Christian and surname completely mirror fashion.

(2) Two or three children at least wrote their names or parts of their names mirror fashion, and then turned over the paper and had another try, but with no better success. It would seem that these children had noticed what they had done, but could not make their left hand do what they wished or expected.

We might, however, supplement the hypothesis under discussion by supposing that there is no real necessity for assuming an established series of muscular sensations, that are then reproduced by the left hand in a "reflected" manner (i.e., mirror fashion). For we can go this far and suggest that quite apart from the establishment of such a muscular feeling there is a "natural" direction of writing with the left hand and that it is from right to left, and that this natural direction may quite well involve a tendency to form letters and even words in a manner the reverse of the natural way with the right; that is to say, that even before letters and words have been copied by the right hand there will be an inclination for the left hand to write mirror fashion unless corrected by visual sensations. Suppose therefore mirror writing is the more natural method of writing with the left hand, but the tendency is corrected by the look of the letters and words, then the first writing, before the correlation of the hand and eye has been perfected, may well be mirror fashion, as has been found in some cases. But such an explanation is negatived by mirror writing with the right hand by beginners.

Again, the original theory as stated by Baldwin might explain in a simple manner why the feeble-minded were more prone than normal children to write mirror fashion with the left hand, after they had learned to write with the right. It would seem quite justifiable to suppose that mirror writing by the hand not habitually used in writing is the automatic reproduction by that hand of the ordinary writing, and that it is due to some mental defect, probably a defect of the higher centres (involving voluntary action), the disarrangement of which has let loose the more automatic "activity of the lower centres, or the nervous arrangements which have escaped injury," and has thus allowed the unpractised hand to follow the more automatic series of movements.[1] In the case of hypnotized "subjects" such an explanation would equally apply, provided the mirror writing was done by the hand not habitually used in writing, but it would of course be negatived by mirror writing with the right hand by right-handed "subjects." There is further evidence in support of this explanation in the case of children who lapse into mirror writing when inattentive. Beeley [6] gives the following examples:—

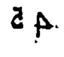
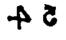
(1) Boy aged 8 years 3 months; bright, lapses into mirror writing when inattentive, knows the difference, very good memory, left-handed writer, left-handed family.

(2) Boy, aged 7 years 11 months: not yet overcome tendency to mirror writing; when his attention shifts from the "executive factor" to the "thought element" lapses to mirror writing. Stated to have written mirror fashion from the beginning, at 4½ years of age, and not to have recognized that it was wrong.

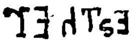
Other experiments were made with letters cut out of stiff paper and placed on the table the reverse side upwards, to see whether mirror writers would arrange them from right to left or attempt to arrange them in the correct direction, i.e., from left to right.

---

[1] If this is a true explanation one would expect mirror writing to be frequently found in the numerous cases of lesions which have affected the higher centres, and have been associated with aphasia and right hemiplegia. But this appears to be very seldom the case among adults; in fact Moutier gives only two examples of mirror writing among all the cases in his book [22] [Nos 361 and 172].

L. J., girl, aged 11 years 10 months: left-handed in all the tests, but writes with the right hand, and wrote mirror fashion with the left at a previous visit. When given the mirrored letters, and asked to form with them the words "cat" and "dog," using her left hand, she arranged them without any hesitation mirror fashion, i.e., from right to left The same letters were then rapidly and without her knowledge turned the right way up; she now arranged them with the same hand in the correct position. Exactly the same result was obtained with the right hand. A 4 and 5 were then given her, and she was asked to make 45 with them, using her left hand. She arranged them thus:  , but seemed dissatisfied with the result. After looking at them for a short time, she took the 5 and placed it on the right side of the 4, thus:  .

The following case seems to indicate that the mirrored letters are sometimes at all events a reproduction of an optical memory image, and not a mechanical reversal of what has just been seen.

E. Y., girl, aged 8 years 1 month · reported to be changing spontaneously from left to right in writing. Left handed in all the tests. As she could not write her name spontaneously it was printed, and she was asked to copy it with her left hand. The name was printed thus. "Esther." She copied it as follows:  —that is, she had changed the small "t" and "e" into capitals "T" and "E."

The curious case of children printing with their right, and writing mirror fashion in a "running" hand with the left, and also those who write mirror fashion slowly and obviously looking at each letter as it is being written, seems to indicate that it is a reproduction of an optical image.

Kerr states: "Similarly infants, tested with copying English and Russian words, only made mistakes with letters common to both alphabets—that is, those with whose forms they had some slight acquaintance—but copied correctly the complicated letters peculiar to Russian, whose form they had never seen before" [16].

From such facts it would seem that something had affected the nerve fibres connecting the hypothetical visual memory of words centre with other parts of the brain; this centre, if there is such a one, is generally believed to be situated in the dominant hemisphere. If this should be the case, the association of left-handedness and mirror writing might be explained by some lesion that has affected the dominant side of the brain, or by a lack of development of this side—resulting in some form of mental deficiency, in a change of right to left-handedness, and in mirror writing.

CHAPTER VI.—DEFECTIVE SPEECH AND LEFT-HANDEDNESS.

Ballard and others [5] [14] have shown that stammering is more prevalent among left-handed children, who write with the right hand, than among "pure" left or right-handed children. Also Lewis [17] has reported that epileptic children—originally right-handed—first hesitated in speech, and then developed a decided stammer, after being trained to write with the left hand in the ordinary way, i.e., in the direction left to right. These same children had previously shown no indication of hesitating in speech, although they had been practising writing with the left hand for two or three months, but the writing had been mirror fashion and from right to left in direction. That is, a change in direction of the writing with the left hand did not seem to affect the speech, but when the direction was unchanged then the speech appeared to be affected. This is also what appears to be the case with some left-hand writers who have changed to the right hand, but of course no experiments have been made with left-hand writers to see whether mirror writing with the right hand would have the same or a different result.

During the present investigation many instances of the same kind were noticed among normal children, but it appeared that among mental defectives there were just as many stammerers among left-handed writers as among left-handed children who wrote with the right hand. In addition to this, defective articulation and other speech defects seemed to be prevalent among mirror writers. No special note was at first made of such defects, as it did not seem probable that the association of mirror writing with defects of the speech was anything more than a coincidence. Later, however, much greater importance was attached to these facts, especially when it seemed likely that many of the children with left-handed activities might be right-handed children (i.e., with a dominant left hemisphere), children who had been driven to the use of the left hand by some defect of the left hemisphere, or the nerve fibres connecting this hemisphere with the right side of the body. As (p. 343) paralysis of the right side (right hemiplegia) is frequently associated with aphasia, and it is therefore possible that with these mental defectives the same defect may have affected not only the hand but also the speech of these children. The following cases are of interest in connection with this subject.

(1) M. G., girl, born March, 1911. Doctor reports: "Meningitis at 2 years; speech defect the most prominent feature; left-handed. February, 1918: ditto, and poor at manual work, October, 1919." She was tested April, 1920: rubs right, throws right, cuts left and right, writes right. The head teacher reports: "She uses her left hand at times in some things."

(2) E. H., girl, born March, 1910. Doctor reports: "Mother says: 'lost her speech for eleven days, and was like an idiot for a week.'"

Tested April, 1920: Throws left, cuts left, picks up left, writes right and left; knits left; dyn., left 13, right 15.

(3) J. P., boy, born February, 1907. Doctor reports: "Premature birth 7 months; drags right leg; ? hemiplegia; talipes; right hand less powerful than left; slight defect of speech; left handed January, 1916; stammers January, 1918; slightly deaf, otorrhœa (right ear); slight paresis of legs."

Tested April, 1920: He still stammers. Throws left, cuts left and right, picks up left, winds left, writes left. This is interesting, as it seems to be a case of slight right hemiplegia with a slight speech defect, which developed into a stammer.

Speech Defects among Mental Defective Children.

	Left-handed		Right-handed	Total
	Write with Left hand	Write with Right hand		
Stammer or stutter	19	20	44	83
Defective articulation..	14	1	18	33
Defective speech, including some cases of defective articulation	39	27	40	106
Lisping associated with other speech defects	6	1	7	14
Great hesitation in speech	0	2	1	3
	78	51	110	239
Percentage ..	32·6 %	21·3 %	46·0 %	99·9 %
	53·9 %			

The above statistics have been made out in connection with children with very defective speech; in every case they have been tested in "handedness." Now, as there are about 81·8 per cent. right-handed among mental defectives, and 18·2 per cent. with many left-handed activities, there are, therefore, four and a half times as many right-handed as left-handed, and if very defective speech is not associated with left-handedness, then there should be among these children with very defective speech four and a half times as many right-handed as left-handed. The results of an investigation on this subject are given above. The numbers are not large, and therefore the result can only be treated as provisional. Cases in which the causes of the defect seem to be clear have been omitted, e.g., split palate, bad adenoids, &c. The number of cases was 239; all the children stammered or had stammered, or were stated to be bad cases of defective articulation or of defective speech.

From the above it will be seen that taking the various speech defects (239), more than half are left-handed, whereas one would expect about 18 per cent. or so to be left-handed unless there was some connection between left-handedness and speech defects. The number of stammerers among the left-handed and among left-handed children who write with the right hand are approximately equal, i.e., nineteen and twenty respectively. These statistics deal exclusively with mental defective children, and it is probable that the same result would not be obtained with normal children. If this is the case, and it seems likely from the results of previous investigators, then it becomes a matter of great importance, as it would seem to indicate that there is something quite distinct in the left-handedness of children under investigation, and that this left-handedness is frequently associated with speech defects.

An interesting case not included in the above is that of a child with defective speech:—

A. W., girl, aged 15 years 8 months: was paralysed (left arm and side) at 2½ years of age. She does everything right-handedly, and cannot even hold a pencil in her left hand. It seemed likely that she was originally left-handed, and that the paralysis had affected her dominant (right) hemisphere, as she is in a mental defective school and her speech is defective. Her mother was therefore questioned, and she stated that "the girl was left-handed until she had the paralytic stroke."

SUMMARY AND CONCLUSIONS.

Left-handedness.

(1) The percentage of left-handed children (i.e., with marked left-handed activities) is much higher in mental defective schools than in ordinary elementary schools:—

Percentage in ordinary schools	7·3
Percentage in mental defective schools	18·2

Note.—(i) About 7·2 per cent. in mental defective schools write habitually with the left hand; (ii) the percentage of left-handed girls (mental defective schools) is about 25 per cent higher than that of the boys.

(2) Left-handedness in mental defective schools is more frequently associated with defects of the speech than is right-handedness (p. 364).

(3) In the case of twins, when one is left-handed and the other right-handed, the left-handed twin is frequently backward, less developed, highly nervous, or even in a mental defective school, whereas the right-handed twin is normal and in an ordinary elementary school. Among twins (a boy and a girl) there is a very high percentage (nearly one-third) where one is left-handed and the other right-handed.

(4) Among normal children the left-handed are frequently the most efficient and capable; among the mental defectives it is exactly the reverse, left-handedness being often associated with marked deficiency. Spontaneous change of left-handedness to right-handedness in writing among mental defectives is often found to be associated with progress in school work and in intelligence.

Mirror Writing.

(5) The percentage of mirror writers is much higher in mental defective schools than in physical defective schools and in ordinary elementary schools.

	Elementary schools	Physical defective schools	Mental defective schools
Percentage of mirror writers	0·48	1·1	8·0

(6) Percentage of mirror writers among left-handed and among right-handed children in mental defective schools:—

	Left-handed	Right-handed
Boys	18 per cent.	1·6 per cent.
Girls	40 ,,	2·8 ,,
Boys and girls	30 ,,	2·2 ,,

(7) Percentage of mirror writers among left-handed children who write with the right hand:—

Boys	32 per cent.
Girls	62 ,,

The great majority of mirror writers are children who are reported to have written with the right and also with the left hand, but this is not invariably the case, as many mirror writers, although left-handed in many things, are said to have never written with the left hand; other mirror writers suffering from right-hemiplegia from an early age have never written with the right hand.

(8) Children who are reported to write on some days with the left and on other days with the right hand can frequently be made to write

mirror fashion by merely asking them to begin at the right edge of the paper. Some of these children write in the same way with the right hand; others cannot be induced to write mirror fashion with the right hand. In many cases these children have never been known to write mirror fashion before. Children reported to have written mirror fashion when young can often be induced to write mirror fashion under similar conditions.

(9) Many left-handed children are reported to have written mirror fashion with the left hand when they first began to write and to have persisted for some time. A few right-handed children also wrote mirror-fashion with the right hand when they first began to write.

(10) In no case has a left-handed writer been reported to have written mirror fashion on changing from left to right hand writing. These same children when asked to write with the left hand frequently do so mirror fashion.

(11) In some instances all the letters are written mirror fashion with the exception of one or two letters, and these are persistently written correctly, and with both hands; in the other cases the correct letter (with left hand) is written mirror fashion with the right.

(12) Of the 109 cases of mirror writing, eight (i.e., 7·8 per cent.) wrote mirror fashion spontaneously with the right hand and correctly with the left.

(13) Mirror writing seems in some cases to be due either to (*a*) a motor memory image, or to (*b*) an optical memory image (p. 361).

Theoretical Conclusions.

There are undoubtedly some peculiar and uncommon characteristics (uncommon even among mental defectives) among some of the left-handed children in mental defective schools, especially among those who write with the right hand. The fact that very many of the mirror writers are left-handed is merely a statement of fact and no explanation. There seems no adequate reason for believing that naturally left-handed children (i.e., with a dominant right hemisphere) should be more prone to mirror writing than the naturally right-handed (i.e., with a dominant left hemisphere), nor is there any reason for supposing that naturally left-handed children should be more inclined to suffer from speech defects than the right-handed. In addition to these facts there are undoubtedly a much larger number of left-handed among mentally defective than among normal children, and many of these are of a very low-grade type.

There seems at present to be only one explanation possible, and this explanation would clear up many, if not all, of the observed peculiarities and apparently contradictory facts. It of course does not go to the root of the matter, and much still requires elucidation.

The hypothesis suggested is that something has occurred which has interfered with the proper functioning of the dominant hemisphere—in the majority of cases described it is, of course, the left hemisphere. Such a supposition would explain :—

(1) Why there are so many left-handed in mental defective schools. The left hemisphere being affected has interfered with the proper functioning of the right hand, perhaps in only a slight degree, as many among the older children change spontaneously from left to the right hand in writing. The same cause has probably affected the functioning of many of the higher intellectual centres, supposed to be situated in the same hemisphere, involving such peculiarities as mirror writing and defective speech.

(2) It would also explain why, in the case of left and right-hand twins, the left-handed twin and not the right-handed is found in these special schools.

Whether the hypothesis is true or only partly true, or whether it is untrue, is not of great moment for the present; it has undoubtedly indicated new lines of investigation, and has been fruitful in suggesting fresh experiments and observations.

BIBLIOGRAPHY.

[1] ALLBUTT'S. "System of Medicine," 1899, vol. viii, p. 3.
[2] ALLEN, E. J. "Mirror Writing," *Brain*, 1896, vol. xix, pp. 385-7.
[3] BALDWIN, J. M. "Mental Development in the Child and in the Race," p. 99.
[4] BALLARD, P. B. "Left-handedness," *School World*, December, 1916.
[5] *Idem.* "Sinistrality and Speech," 1912.
[6] BEELEY, A. L. "Experimental Study of Left-handedness," Supplementary Educational Monographs, University of Chicago, June, 1918, Chicago, 1918.
[7] FULLER, J. K. "Psychology and Physiology of Mirror Writing," University of California, published in *Psychology*, May 8, 1916, vol. ii, No. 3, p. 199.
[8] GALTON, F. "Inquiries into Human Faculty," 1911, p. 156.
[9] GOLDSCHEIDER. *Arch. f. Psychiatr.*, 1892, Bd. xxiv, S. 503.
[10] IOTEYKO, JOSEFA. "The Science of Labour and its Organization," 1919, pp. 96, 126, 143.
[11] IRELAND, W. W. *Brain*, 1881-82, vol. iv, p. 361.
[12] *Idem.* *Brain*, 1880-81, vol. iii, p. 207.
[13] JACKSON, J. "Ambidexterity," pp. 10, 141, 163.
[14] JONES, FRANKLIN. "A Study of Handedness," University of S. Dakota, series xvii, March, 1918, No. 14.
[15] JONES, H. MACNAUGHTON. "Ambidexterity and Culture," p. 66.

[16] Kerr, James, M.D. "Left-handedness and Mirrored Writing," *School Hygiene*, 1920, vol. xi, p. 39.

[17] Lewis, A. E. "Left-side and Ambidexterity," *Times Educational Supplement*, May 6, 1920.

[18] Lochte. *Arch. f. Psychiat.*, 1896, Bd. xxviii, S. 379.

[19] Lombroso. *North American Review*, 1903, vol. clxxvii, p. 440

[20] McHattie. "L.C C. Report of Council," *Public Health*, 1913, vol. iii, p. 120.

[21] Monroe. "Mirror Writing", *Cyclopædia of Education*, 1913, pp. 108, 252.

[22] Moutier, F. "L'Aphasie de Broca," 1908, p. 118

[23] Schafer. *Berl. klin. Wochenschr.*, 1911, Bd. xlviii, S. 295.

[24] Sherlock, F. B. "The Feeble-minded," 1911, p. 86.

[25] Storck. "Les Stigmates anatomiques et physiologiques de la dégénérescence et les pseudo-stigmates anatomiques et physiologiques de la criminalité," Lyon, 1902, p. 97.

[26] Wallin, J. W. W. Annual Report, Board of Education, St. Louis, 1915-16.

[27] Weber. *Zeitschr. f. Klin. Med.*, 1895, Bd. xxvii, S. 260.

[28] Whipple, A. M. "Manual of Mental and Physical Tests," 1916, part i, pp. 102, 107, 139.

TONUS AND THE REFLEXES.

BY DR. J. K. A. WERTHEIM SALOMONSON.

Professor of Neurology in the University of Amsterdam.

[*Delivered before the University of London on January* 17, 1921.]

THE Anglo-Batavian Society put forward the project recently for a series of lectures to be delivered in England by Dutch and in Holland by English medical men. Through the whole-hearted co-operation of the Royal Society of Medicine and the University of London, as well as of the Royal Academy of Amsterdam, this project has become reality. I have been invited to inaugurate this series from the Dutch side. The undoubted temerity of accepting this invitation is only to be explained by my knowledge of the generous judgment and courteous forbearance with which my fellow-workers in the field of science are received in your midst. Yet I fear that I shall have to crave your kindness and indulgence.

The subject of my lecture is one which in Holland is engaging many workers, and on which I have also expended considerable thought and time as well. I mean the question of tonus and reflexes. In my work on this subject I made an extensive use of Einthoven's ingenious invention, the string-galvanometer. I hope to show you a very small part of this work and to submit to your consideration some remarks on the result and conclusions to be drawn from it.

The investigation of the electric response of contracting striped muscle by means of the string-galvanometer has led us to classify the muscle contractions into three different groups. A representative of the first group may be found in the simple muscle twitch, such as is elicited by stimulating a motor nerve of the muscle itself with one condenser discharge or an isolated break-shock from the secondary of an induction coil. The resulting muscle twitch is characterized by its short duration as well as by its being preceded by a double-phase action current. During the contraction itself the muscle is electrically inert. An example of the second group is the voluntary muscle contraction, which is characterized by a current of action in the form of a rather irregular alternating current with a frequency of about 50 periods per second. The electric responses of the first and of the second kind can easily be recorded in our patients. The last group comprises all kinds of muscular actions in which no electric current of action can be detected.

To these three groups we might possibly add a fourth, comprising contractions caused by special forms of electric stimulation, in which we are able to record a few abnormal forms of electrical response. As these contractions are always artificial and never occur physiologically, and as they have no direct bearing upon our subject, we need not take them into further account.

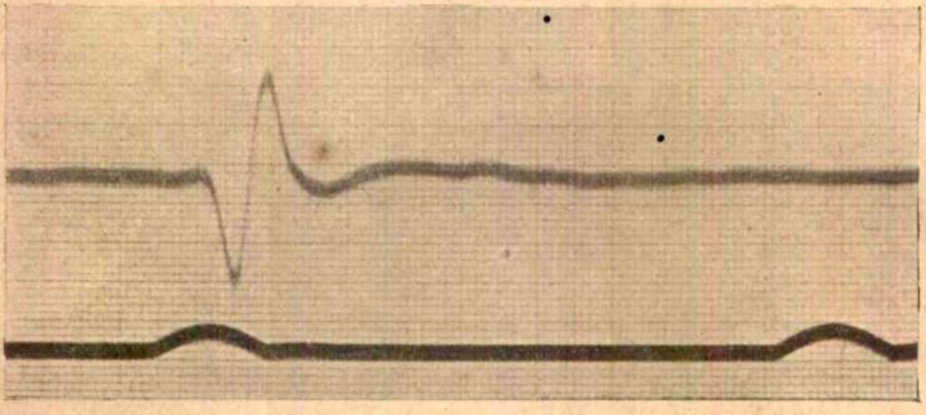


FIG. 1.—Knee reflex. Electromyogram. 400 mm. per second.

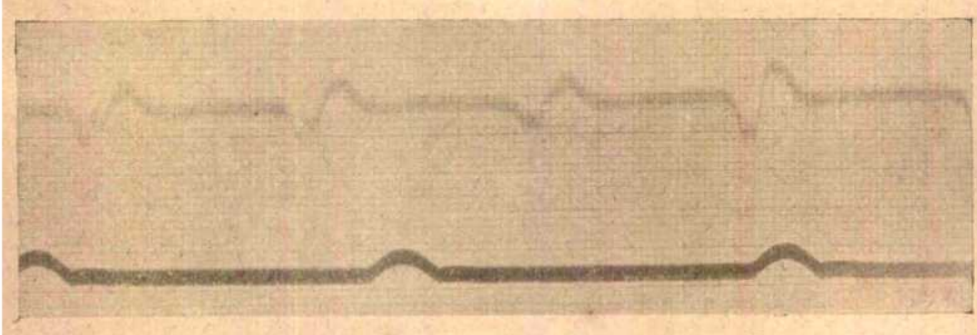


FIG. 2.—Knee clonus in myelitis. 250 mm. per second.

In human physiology and pathology muscle contractions of the first kind, in the form of simple muscle twitches, are to be found, of course, after electric stimulation, but also, as I found in 1907, in all deep reflexes, tendon reflexes as well as periosteal reflexes. In cases of muscle-clonus in lesions of the lateral tracts, I found a regular series of simple twitches, each starting after a complete biphasic action current. Between these the galvanometer-string practically does not move at all. I have not been able to find a single other instance of the occurrence of simple muscle twitches under physiological or pathological conditions.

The second kind of muscle contraction is met with in every voluntary movement, as was first demonstrated by Piper. The irregular alternating current of some fifty periods per second is the electric phenomenon which is typical of the tetanic contraction. If found in any contraction we may be absolutely sure that we have a tetanus before us. In cerebral contraction, such as epileptic convulsions, the string-galvanometer again shows the tetanic character of the muscular shortening. In two cases of myoclonic epilepsy I at first thought that I had at last found single muscle twitches of cerebral origin, but a closer examination of the

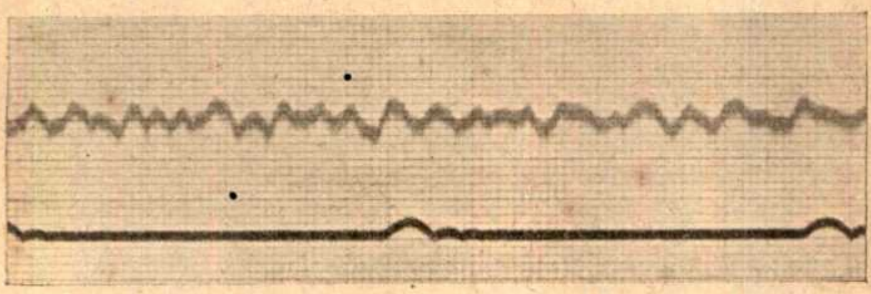


FIG. 3.—Voluntary tetanus. Time marks 0·2 second.

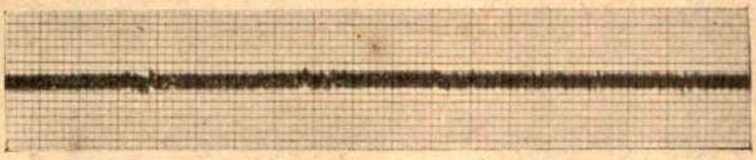


FIG. 4.—Myoclonic-epilepsy. Very short tetanus, nearly resembling simple twitches.

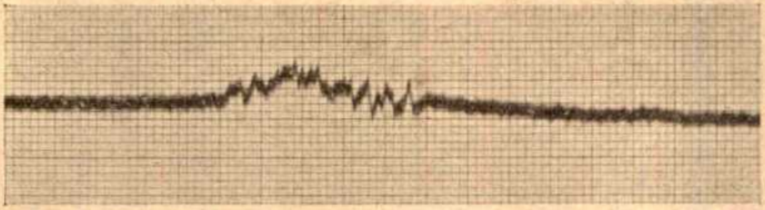


FIG. 5.—Electromyogram in normal plantar reflex. 100 mm. per second.

galvanometric records proved them to be also short tetani, the shortest I have ever seen. Many other different kinds of muscular action belong to this group, as the examination with the string-galvanometer proves. Such is the case with all cutaneous reflexes, as I had found many years ago. The duration of the tetanus may be very short, as short as 0·1 second, though generally it is much longer, yet we never fail to find the irregular oscillations of the string which indicate that the contraction was a real tetanus.

The third kind of muscle action is perhaps the most interesting. We may observe strong muscle spasms and also notable changes of the muscle tension without any electric action current. All changes in muscle tonus occur in this way and do not seem to cause any change in the electric state of the muscle. If the triceps brachii be connected to the galvanometer-terminals and the arms passively extended or bent, the string does not move. Yet the passive movement of extension is accompanied by a shortening of the muscle, the tonus-contraction. Similarly a tonus-relaxation does not act upon the string. In the case of myelitis with complete paralysis of the lower limbs, we may find enormous muscle spasms which do not show any action current. The same may occur in hemiplegia, lateral sclerosis of the cord, multiple sclerosis or generally in complete degeneration of the pyramidal tracts. I found them also in focal disease of the corpus striatum and regio subthalamica. In all these morbid conditions we have strongly hypertonic muscles. As far as we can judge, the absence of a typical action current seems to be the one and only means of differentiating between a tetanic and a tonic spasm or contraction. We should therefore use the Einthoven instrument in order to discover the nature of any muscle tension about which we are in doubt. Fröhlich and Meyer employed it for this purpose with a patient suffering from tetanus infection and found that the muscle spasms were tonic and not tetanic. I had occasion to examine a cat infected with tetanus by Professor Magnus, and easily obtained a record from the spastic triceps muscle showing a straight line interrupted only by small regular notches produced by the action current of the heart. On the other hand I found with Dusser de Barenne decerebrate rigidity to be a tetanic state. In veratrine poisoning we get a tonic muscle spasm, whilst strychnine poisoning produced real tetani, as was discovered years ago by Lovén and by Burdon Sanderson. Cataleptic muscle spasm is described by Fröhlich and Meyer as being of tonic origin, which does not seem to be in contradiction to an earlier investigation by Gregor and Schilder, whose curves showed slight irregularities. As regards tetany in children and adults we have as yet no certain data. Two infantile cases which I examined did not yield satisfactory results and left me undecided as to the origin of carpopedal spasms. Yet I incline to the belief that these spasms are of tonic origin.

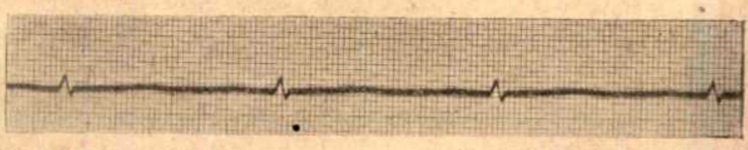


FIG. 6.—Tetanus infection. Electromyogram of triceps. 50 mm. per second.

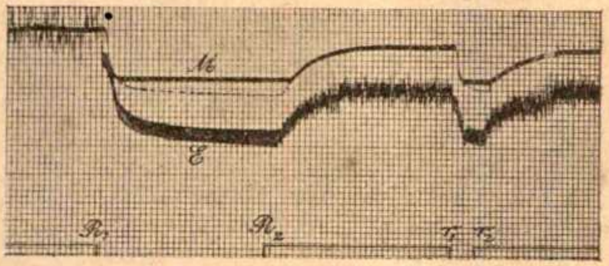


FIG. 7.—Decerebrate rigidity. M = Myogram. E = Electrogram. From R_1—R_2 stimulation of central nerve stump. After Einthoven.

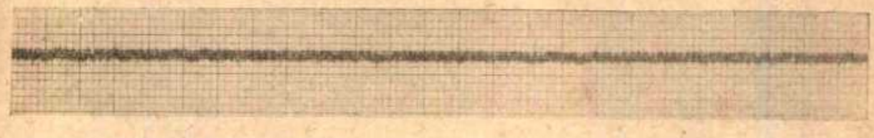


FIG. 8.—Infantile tetany. Action current in flexors of fingers. 100 mm. per second.

The question about the after-contraction in Thomsen's disease seems likewise to have been settled by Gregor and Schilder, who were not able to find the action-current typical of tetanus. I too have failed to find it, and therefore cannot agree with the recently published opinion of Schäffer who, after cessation of the voluntary tetanus, still found slight indications of an irregular oscillatory action-current which gradually tend to subside. I believe that these movements of the string are not caused by the tonically contracting muscles, but by the active contraction of the antagonists, which contract involuntarily to oppose the after-contraction.

An exceedingly curious kind of after-contraction was first described under the name of "Katatonus-Versuch" by Kohnstamm in the *Neurologisches Zentralblatt* of 1915, but, as far as I know, has not yet penetrated into English medical literature. We can elicit this after-contraction in different muscles, but most easily in the deltoid. To do this, we should first voluntarily cause the muscle to contract isometrically with as much force as possible, and keep this up for perhaps a minute, the muscle being kept stretched all the time by fixing the arm. If then the voluntary impulse be stopped and at the same time the arm be released, we feel that the arm is drawn upwards, without, and even against, our will. Of course, we can easily prevent the arm being lifted, but if we try to remain absolutely passive, refrain from any voluntary impulse and just let the arm do what it seemingly wishes to do, it is lifted. The experiment succeeds equally well with the hand extensors, whereas it is far less pronounced with most of the other muscles of the body. Csiky has shown that a strong prolonged faradic tetanization, during which the muscles are prevented from shortening, will also cause this after-contraction to appear. The duration of the after-contraction may be from one to ten seconds. This katatonus experiment has been ascribed to various causes. Kohnstamm thinks it may be an involuntary tetanus, whereas Csiky regards it as an idiomuscular contraction. If by this he means the same thing as what I call a tonus-contraction, I can agree with him, for examination with the string-galvanometer shows it to be an undoubted tonus-contraction.

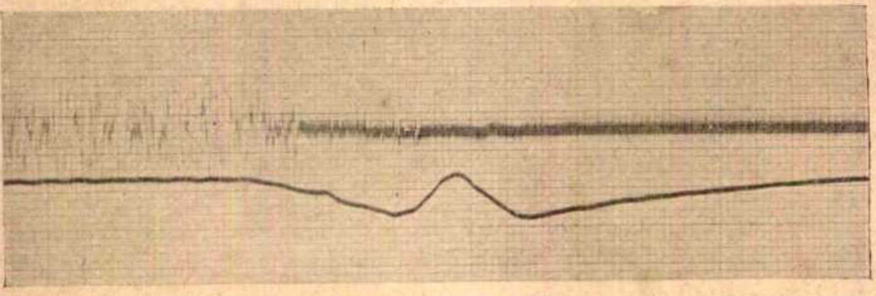


FIG. 9.—Katatonus experiment. Lower curve mechanical myogram. 50 mm. per second.

There are many forms of hyperkinesis, regarding which we do not yet know whether they ought to be considered as tetani or tonus spasms. I shall not discuss these here, but shall give you later on the results I obtained in one particular field of research, namely, the reflexes. I have already mentioned that in normal deep reflexes we always get a simple muscle twitch preceded by a diphasic action current, and that the knee and ankle clonus as well as the hand clonus in cases of sclerosis of the lateral tracts is composed of a regular series of simple twitches, each giving one single diphasic action current. Between each two successive electric impulses the muscle is electrically inert. But how is it with hysterical clonus? Here the galvanometric record shows the presence of a typical tetanus on which is superposed a series of somewhat stronger and probably not entirely regular diphasic action currents. It is generally assumed that the mechanical condition for the appearance of clonus demands an increased reflex-irritability and at the same time the presence of a certain amount of muscle-tension. Only the co-existence of both circumstances renders it possible for the muscle to contract clonically. With degeneration of the lateral columns this increased muscle-tension is caused by increased tonus, whereas in hysterical clonus this is due to a tetanic muscular contraction. Although in general we shall not have any great difficulty with the differential diagnosis between organic and hysterical clonus, we may with advantage use the string-galvanometer for difficult or doubtful cases. We may probably even go a step further and say that the symptom here mentioned constitutes the only absolute and reliable symptomatical and genetic difference between the two conditions.

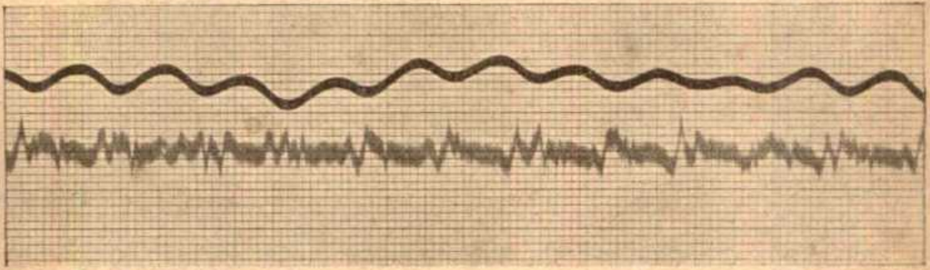


FIG. 10.—Hysterical knee clonus. 100 mm. per second.

Frequently much definite and useful knowledge may be gained by simply recording the mechanical myogram in deep reflexes, especially when they are exaggerated. The normal knee-jerk gives a myogram which may best be compared to one from a gastrocnemius preparation of the frog. But as soon as we have to deal with exaggerated reflexes we hardly ever find this simple form. More often we find clonic contractions either in a short or in a longer series, each of the components giving the well-known biphasic action current, in the same way as with the organic knee-clonus. In another group of cases we find that the initial twitch is followed by only one more contraction, which may commence after the principal jerk is entirely finished, but generally a little earlier. This depends upon the duration of the first twitch, which generally lasts from 0·2 to 0·3 second, the second contraction usually beginning 0·25 to 0·3 second after the moment of percussion. Now, it is curious to notice that these double contractions may occur under widely different circumstances. I shall give a few instances.

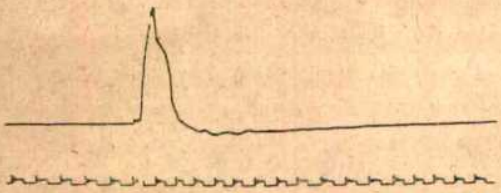


FIG. 11.—Knee reflex, simplest form.

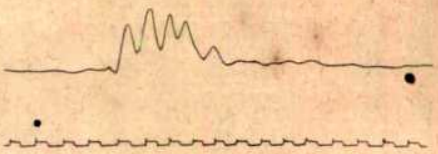


FIG. 12.—Knee reflex, simple clonic response.

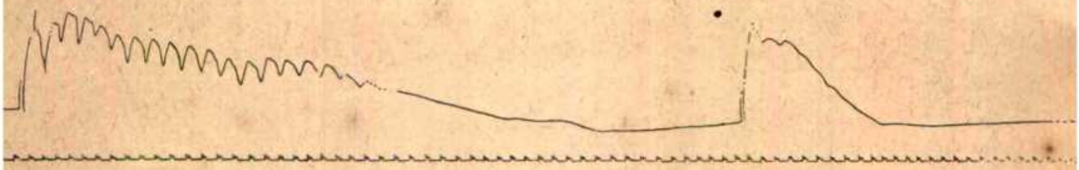


FIG. 13.—Knee reflex, clonic after-contractions superposed on tonic secondary contraction.

We frequently find a double contraction in cases of chorea minor where its occurrence has already been described by Gordon Hey, Eshner, Kleist, generally as a lengthened response. This symptom was considered as a choreic movement following the reflex extension. The graphic record proves that the muscle invariably relaxes at least partially between the two contractions, and furthermore that the last regularly occurs after a definite latent period of 0·21 to 0·25 second. Its duration is not constant. Fahrenkamp has examined the action current and found the second twitch to be a tetanus. I found the same thing, so that we may take it as a fact that the knee-jerk is sometimes immediately followed by a second contraction of tetanic character. In several cases of sclerosis of the pyramidal tract the mechanical myogram of the knee-jerk also showed a double contraction not infrequently of different types.

In one kind the second contraction was an unbroken one of very short duration, although contractions lasting some 10 to 20 seconds may also be met with. In another type it appeared in the form of a series of clonic contractions from anything between two or three and a few hundred. Whereas this last clonic group was found to be electrically defined as an organic clonus, the first group did not give any deflection of the string. This implies that the knee-jerk was followed by a longer or shorter tonus-contraction. But a point of interest is that we are able to produce at will in some patients either a tonic or a clonic after-contraction. We do this by only slightly modifying the way in which we support the knee of the patient; if necessary we can easily get

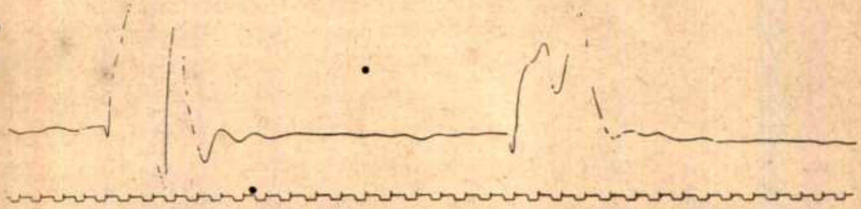


FIG. 14.—Knee-reflex in chorea. Double contraction.

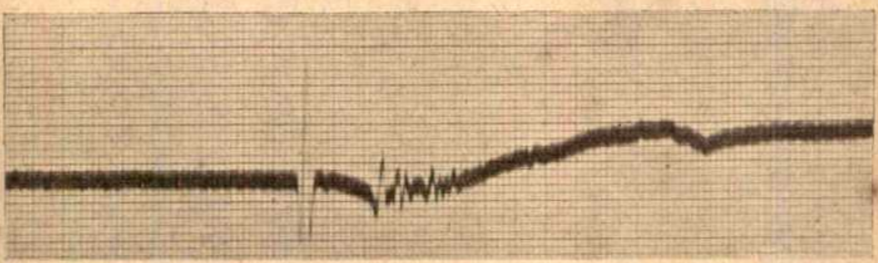


FIG. 15.—Knee reflex in chorea: Gordon Hey type. Action current of quadriceps. 100 mm. per second. Simple twitch followed by tetanus.

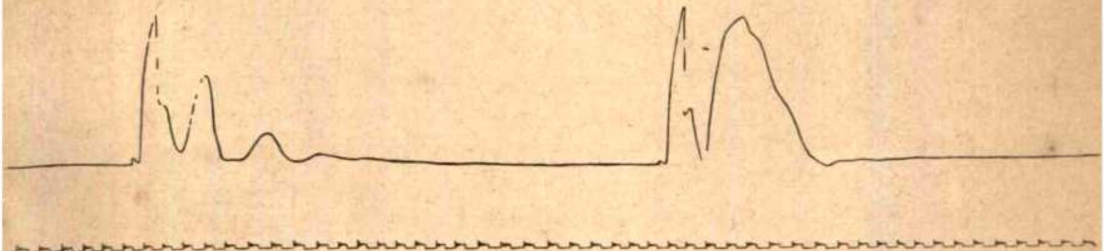


FIG. 16.—Knee-reflex in disseminated sclerosis. Double contraction.

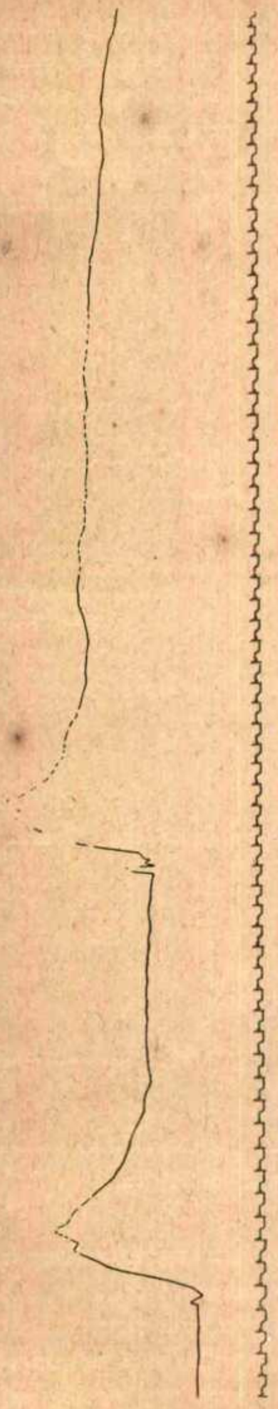


FIG. 17.—Tonic after-contraction of long duration in cerebral pseudobulbar paralysis.

alternately tonic and clonic after-contractions. This may be explained by first looking at the myograms. We see in both a tonic after-contraction. Now in order to get a clonus we have only to render the conditions for it as favourable as possible. If the upper part of the leg be immovably fixed the increase of tension in the quadriceps caused by the tonic after-contraction is sufficient to allow of the occurrence of a clonus. But if we allow the leg to be nearly fully extended during the after-contraction the tension does not rise so high as to permit of a clonus. The same is the case if the knee is bent much farther than is usual whilst provoking the knee-jerk.

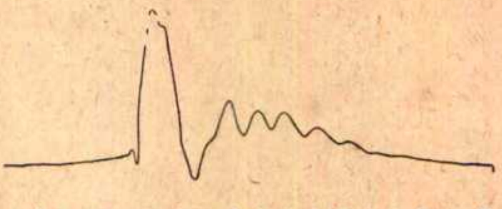


FIG. 18.—Knee-reflex, clonic after-contraction.

Muscle cut off from the central nervous system loses its tone. The normal maintenance as well as any variation of muscular tonus is a reflex action. The well-known Brondgeest experiment has probably been the first direct proof. As our knowledge of tonus is still far from complete, many have endeavoured to obtain a better insight into tone mechanism. Amongst those who have investigated the mechanical properties of normally innervated muscle substance as compared with those of muscle-protoplasm without tonus innervation, I must mention Langelaan of Amsterdam, who has contributed a valuable paper on this subject in *Brain*. Professor Pekelharing in Utrecht was the first to demonstrate the chemical difference between ordinary contraction and tonus contraction. It is by the last that the creatine production is principally governed. De Boer has investigated the peripheral reflex mechanism itself and demonstrated that the centrifugal path probably lies along sympathetic nerve-fibres. Lastly, Boeke of Utrecht has found in all striped muscles new motor nerve-endings, which are not in connexion with the motor ganglion cells of the anterior grey substance in the cord. Personally I incline to the belief that Boeke's fibres, which undoubtedly are of sympathetic origin, conduct the centrifugal tonus impulses, though I am aware that Magnus and Dusser de Barenne do not share this opinion.

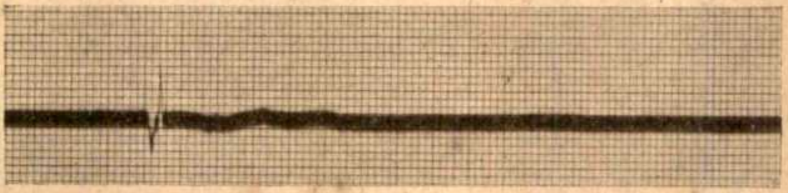


FIG. 19.—Electromyogram in knee-reflex with tonic after-contractions. Same case as fig. 17.

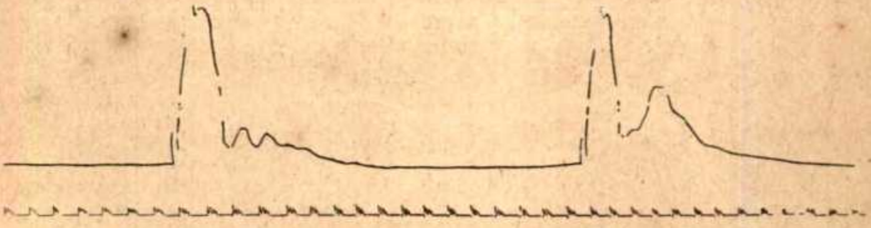


FIG. 20.—Knee-reflex with clonic and with tonic after-contraction.

The afferent reflex fibres, bearing the impulses from the muscle substance, pass through the posterior roots into the spinal cord, where they terminate in the lateral horns. From there a connector fibre passes through the anterior root, through the grey communicating rami into the sympathetic ganglion, where a third sympathetic neuron begins and returns to the motor nerve, where it runs parallel with the motor fibres to the muscle substance. Here it terminates in Boeke's end-organs. De Boer cut the grey rami communicantes in a frog and found the tonus to be diminished at the same side. A few days afterwards the muscle was stimulated by a single break shock of the induction coil. When the stimulus was applied to the spinal centre the form of the muscle twitch was not the same as when the muscle was directly stimulated. In the former case the duration was shorter and the myogram showed the simplest and best known form. With direct stimulation the curve exhibited a secondary summit following the first one. De Boer considers this second elevation, generally known as "Funke's nose," as a tonus-contraction caused by direct stimulation of tonus substance, at the same time as the anisotropous muscle substance is stimulated and contracts. This tonus substance can no longer be stimulated from the spinal cord after degeneration of the sympathetic fibres.

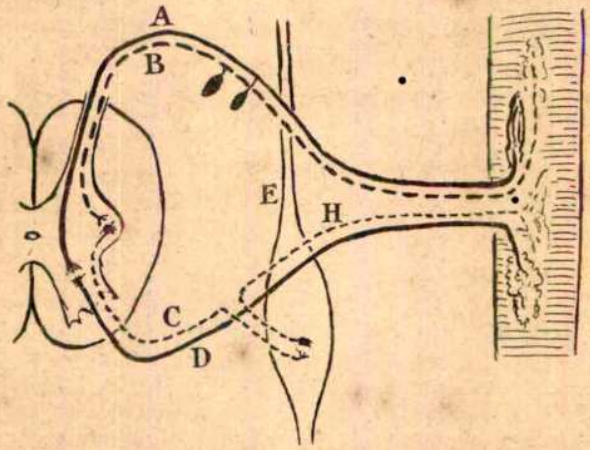


FIG. 21.—A and B, peripheral sensory neuron ; C, peripheral connector neuron ; D, motor neuron ; H, sympathetic neuron ; E, sympathetic nerve.

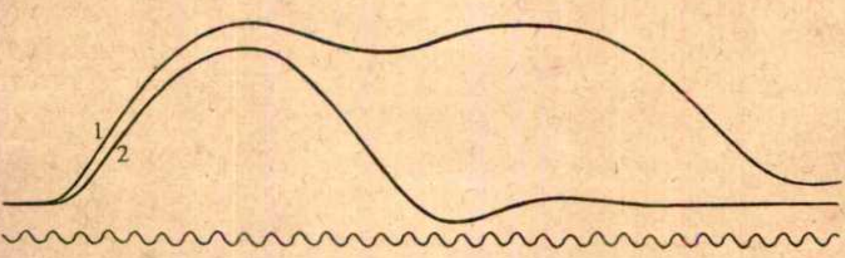


FIG. 22.—1, Contraction of gastrocnemius after direct stimulation of the muscle. 2, *Idem* after stimulation of the spinal cord, the rami communicantes having been cut five days before.

In the myogram of human muscles we practically always find the elevation of Funke; in hypertonic muscles the elevation is more plainly visible, as was clearly demonstrated by curves published by Mendelsohn of Paris and Kollarits of Budapest. Both describe a spasmodic muscle contraction in which the tonus elevation dominates nearly the whole curve. We have seen the same thing in curves of the knee-jerks. We may therefore state that the knee-reflex is not so simple after all, but yet that it contains in itself the cause of a tonus-reflex. As this is also true of all the other deep reflexes we may say that a tendon-reflex is really composed of two superposed reflexes.

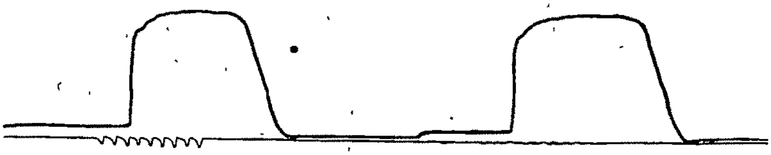


FIG. 23.—Tetani with sixteen and eighteen impulses per second; healthy man.

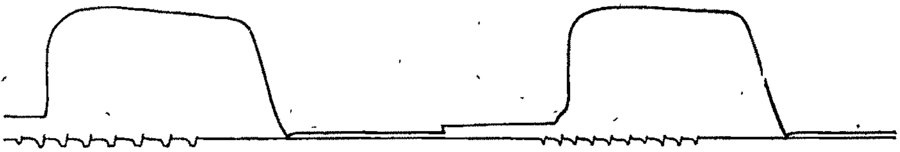


FIG. 24.—Tetani with ten and fourteen impulses per second. Disseminated sclerosis.

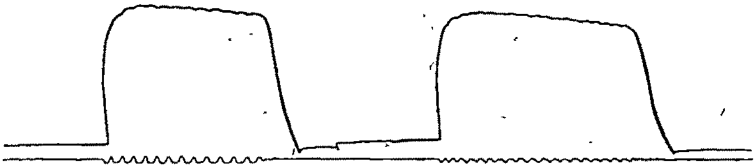


FIG. 25.—Tabes dorsalis. Tetani with 22 and with 24 impulses per second.

I can state here that the degree of tonus in human muscles has a considerable influence on the facility with which a complete tetanus may be provoked by faradic currents. Ratu Langi and I found that whilst in normal muscles about 18 interruptions per second of the induction coil were needed to elicit a complete tetanic contraction, hypertonic muscles gave a complete tetanus even with 10 or 12 interruptions per second. On the other hand, not less than 22 to 24 interruptions per second were needed with patients with locomotor ataxia whose muscles were quite atonic.

Let us now return to the patients with chorea minor in which the knee-reflex proper is followed by a shorter tetanic contraction. I have already remarked that this second part has a definite latency. This is one of the reasons why I favour the opinion that we must not consider this tetanus as a voluntary contraction, but should rather regard it as a reflex contraction. This would imply that the knee-jerk has a third component—viz., a tetanic one. There are two other facts which seem to point to the same thing. In a few rare cases of tabes dorsalis or of chronic peripheral polyneuritis we find, on trying to elicit a knee-jerk, that the quadriceps contracts, but with a notably lengthened latency. This fact was observed and investigated first by Westphal, and is generally known as the pseudo-knee-jerk. Westphal regards this as a cutaneous reflex, and in some eight or ten cases in which I have seen this pseudo-patella-reflex I always found a constant though great latency of the order of 0·18 to 0·2 second. The other fact is that with many nervous persons the tapping of the patella tendon does not elicit a simple twitch but a jerk, immediately followed by convulsive extension of the whole lower limb. We can best explain these three different varieties of the knee tendon-reflex by assuming that not only a medullary centre but also a cerebral centre is provided for the knee-reflex. The reflexogenous zone for the central component extends over the part of the skin nearest to the patellar tendon. The centre itself is a secondary one of but little general use, and becomes active only under favourable circumstances, such as are found in patients suffering from chorea minor, or in highly-strung nervous persons, where either its excitability is increased or voluntary inhibition is diminished, or in rare cases of locomotor ataxia or polyneuritis, where it acts vicariously for the medullary knee-reflex centre. In character this cerebral knee-jerk is a skin-reflex with tetanic response. As the ordinary knee-jerk, which it should sustain and strengthen if necessary, is generally quite adequate and sufficient for its purpose, the action of the cerebral part of the reflex is hardly ever called for. Even when this might happen voluntary inhibition will generally prevent its activity, just as might happen in a minor degree with some ordinary inconstant skin-reflexes. Though this explanation permits of viewing a few widely different subjects from the same standpoint, I should have hesitated to accept it were it not that I had found the same thing with another group of reflexes which I described a few years ago under the name of shortening reflexes. If in some patients the relaxed foot is passively bent with a quick movement, so as to bring the ends of the tibialis anticus muscle nearer together, we sometimes observe a short contraction of this muscle. In the same way a passing contraction may be provoked in the semimembranosus, semitendinosus and biceps, if the knee is passively bent somewhat rapidly. These contractions appear when the muscle is shortened, which proved to be the reflexogenous moment. Hence the name. The latency of these reflexes is extremely short: of the order of 0·04 second, which places them on a line with

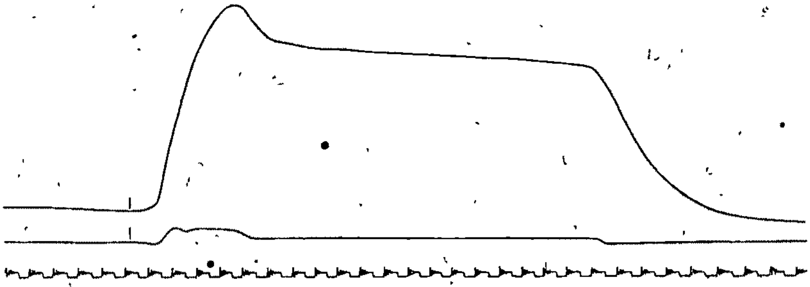


FIG. 26.—Shortening reflex in the tibialis anticus of a healthy man. Upper curve: passive displacement of the foot. Middle curve: contraction of tibialis anticus. Time marks of 0·1 second.

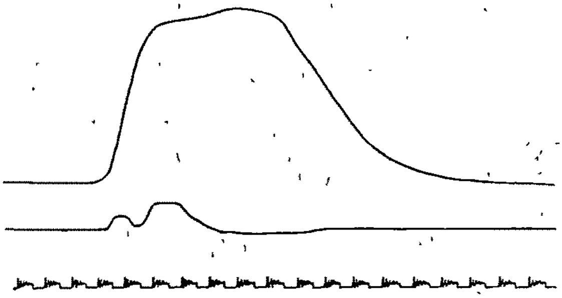


FIG. 27.—Shortening reflex of the healthy leg of a hemiplegic patient.

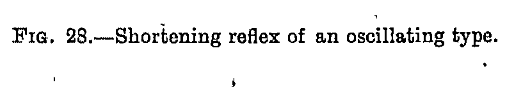


FIG. 28.—Shortening reflex of an oscillating type.

the tendon-reflexes. They also show the same peculiarities as the latter, being for instance accompanied by a tonus variation to which the muscle twitch forms the introduction. In a few patients I was able to record double contractions, as with the Gordon-Hey reflex. Rarely was a clonic after-contraction observed. These reflexes fail to appear whenever the tendon-reflexes are absent. But if these last are exaggerated, the same does not happen with the shortening reflexes. Curiously enough we find them generally easy to provoke in hemiplegic patients, not on the paralysed side but in the other leg. These reflexes show the same complexity as tendon-reflexes: first the tonus variation, and often the secondary tetanic component, showing them to have a cerebral centre too. If the activity of the cerebral centre be greatly increased the shortening reflex may change into Westphal's paradoxal contraction. I consider this last as a pathological variety of the shortening reflex if we except those cases in which it is of a cataleptic origin.

We have now to consider the cutaneous reflexes. These too I believe to be more complicated in the matter of the centre than we are taught in the text-books. This seems to be absolutely certain with respect to the defence reflexes from the skin, which generally possess a medullary and a cerebral centre. In animals the spinal centre is probably the more important one, whereas in man it is subordinate to the cerebral centre, which is generally located in the cortex. If this be destroyed the skin reflexes disappear, but usually reappear after a fairly long period and more often in slightly modified form or intensity. In several patients with section of the spinal cord where the post-mortem microscopical examination proved the section to be a complete one, I have seen the skin-reflexes return after an absence of six months (e.g., Brouwer). We may, and indeed ought to, assume a double centre for the skin-reflexes as well as for the deep reflexes, the difference being that in the cutaneous reflexes the more important activity belongs to the cerebral centre, whereas in the deep reflexes this is only a secondary centre of minor importance. That a tonus reflex also forms part of any skin-reflex need hardly be mentioned, as we cannot imagine a tetanic contraction without tonus reflex. This last is a positive one in the contracting muscles, a negative one in the antagonists.

In a few reflexes the complexity is still more pronounced than would appear from our discussion. The best example is perhaps the plantar reflex. In children who cannot yet stand or walk the plantar reflex, obtained by stroking the lateral side of the sole of the foot, is a pure protective reflex. The knee is bent, the foot is dorsally extended, in which also the long extensor of the great toe assists; frequently the whole leg is drawn up. As far as the great toe, it shows the reflex type described by Babinski. But as soon as the child is able to walk a new reflex appears, in which the toes are flexed, as soon as the sole of the foot is stroked or pressed. Kalischer first pointed out that this new reflex is of great teleological importance. It causes the toes to be pressed against the floor as soon as the sole of the foot touches it, making the surface of contact as large as possible and rendering the conditions for standing or walking and for keeping the equilibrium as favourable as they can be made. Now the old extension reflex has not disappeared; it still exists but is invisible on account of the presence of the new and stronger reflex. The strength of the new reflex is probably to be found in the strength of the contracting muscles, as the toe flexors are stronger than the extensors in physiological condition. Consequently the preponderance of the action of the flexors over the extensor muscles practically renders the escape reflex unnoticeable. If the pyramidal tract has degenerated the toe flexion reflex disappears, only to leave the extension reflex of the Babinski type. How is this fact to be explained? Babinski, Kalischer and others speak of a disturbance of the physiological preponderance of the flexor muscles. But why should in pathological conditions the flexion be weaker than the extension if, normally, the extension was weaker? How can destruction of the lateral tracts cause this change? My answer to the question was that the explanation must not be looked for in the muscles themselves but in the behaviour of the different reflex-centres. Before giving a fuller exposition of my meaning, I may be allowed to say a few words on the muscle action in the Babinski reflex.

A mechanical record of the contraction of the extensor hallucis longus during a Babinski reflex shows that contraction is always a long one, somewhat resembling the slow contraction in reaction of degeneration, but mostly of still longer duration. If an electro-myogram be taken at the same time we can see better what happens. In this record on a plate moving 50 millimetres a second the electro-myogram shows an irregular alternating current lasting 0·66 second. About 0·14 second after the commencement of the action current the mechanical response begins, very gradually at first and only after 0·36 second very rapidly. The whole mechanical response takes not less than 2·7 seconds. In another record we see nearly the same thing. The time between the commencement of the electrical and the mechanical response is again about 0·12 second. The duration of the action current, or—what is the same thing—of the purely tetanic part of the contraction, happens again to be 0·70 second, whereas the whole contraction takes over 3 seconds. In yet another electro-myogram the tetanus took only 0·11 second, though the total contraction lasted rather more than 2 seconds. These records prove that in the Babinski extension reflex, too, the tetanus is the beginning of the reflex which is immediately followed by a tonus-contraction. But they also show that the tetanus, which is the real primary reflex, is not sufficient in itself to cause more than a hardly perceptible movement of the hallux; it is the tonus variation that causes the

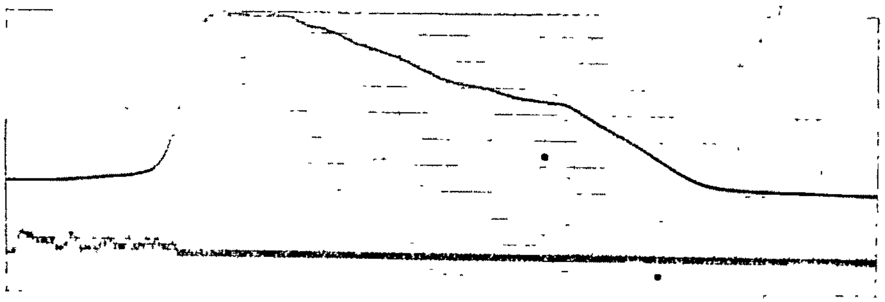


FIG. 29.—Babinski reflex. Myogram and electromyogram. 50 mm. per second.

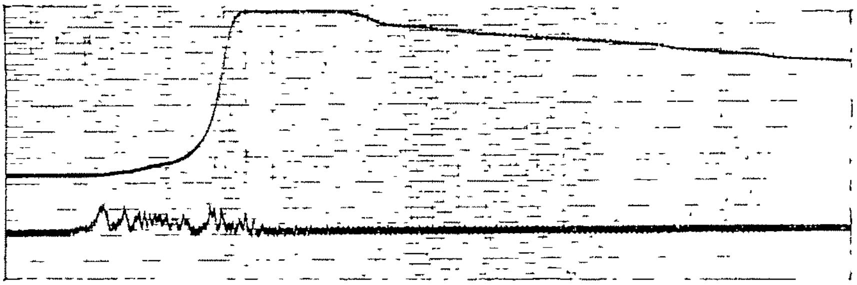


FIG. 30 —Babinski reflex. Myogram and electromyogram. 50 mm. per second.

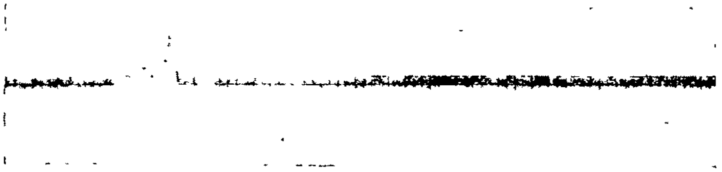


FIG. 31.—Babinski reflex. Electromyogram. 10 mm. per second.

extensive movement. I need scarcely add here, that I never succeeded in getting the tonus-contraction without the tetanus. Clinical examination of the patients from whom these records were taken proved that the tonus of the muscle of the hallux during rest was not greatly changed; a passive flexion or extension of the hallux met with almost the same rather small resistance. Apparently it was not the tonus itself which was exaggerated, but the irritability of the tonus-reflex mechanism. With normal muscles we never find anything of this kind. As soon as the action current disappears the contraction ceases. Only in muscles very nearly exhausted—as we have already explained at some length when discussing the katatonus experiments—do we get after-contractions of tonic origin. Consequently there can hardly be any doubt about the interpretation of these curves.

Before returning to the question of the plantar reflex, I must point out that the centres of the phylogenetically old reflexes are situated in the spinal cord. The younger a reflex is phylogenetically, the higher up in the cerebro-spinal system must we look for its centre. Where two or more centres are present we may expect that the oldest centre will be found in the lower part of the central nervous system, generally in the spinal cord.

Let us now return to the plantar reflex. The reflexogenous zone is the sole of the foot. As part of the integument it possesses in the first place the property, belonging to any part of the skin, of being the reflexogenous zone for a protective reflex. This is a phylogenetically very early reflex with a low spinal centre. In man this spinal reflex is not adequate and it has to be assisted and restrained by cerebral impulses—involuntary as well as conscious ones—undoubtedly in a far higher degree than in animals. But this means the development of a cerebral centre which gains in importance the higher up we look in the animal series, till we arrive at man, where it has attained far greater importance than the original spinal part of the reflex.

The sole of the foot is at the same time the reflexogenous zone for the toe-flexion movement, the reflex needed for walking and standing. This reflex develops only as the child learns to stand and walk, and we may even say that the act of learning to stand is the same thing as the gradual acquisition of the reflex. As standing and walking only take place after a cerebral impulse and are only possible by continuous cerebral impulses, the whole mechanism of these actions comes very near to being a complete cerebral automatism. As soon as the cerebrum directs the foot to touch the floor toe-flexion appears as the consequence of a phylogenetically late and consequently cerebral reflex. Now we also see why teleologically the toe-flexion reflex should be stronger than the protective toe-extension reflex in normal man. If this were not so, walking would be extremely difficult, not to say impossible, as with each step the toes would be lifted from the floor instead of being spread so as to enlarge our contact with the earth. That is the reason why the act of learning to walk is largely synonymous with the acquisition of this reflex. Even the age of the child is immaterial; in rare cases where children learn to walk at a more advanced age, as in late rickets, the original Babinski type of the plantar reflex persists till the child is able to walk. But as soon as this happens the flexion type of the plantar-reflex is provoked by touching the floor with the sole of the foot as well as by striking it.

Starting from these considerations we arrive at a plausible explanation of the Babinski reflex in lateral sclerosis. As the centrifugal impulses for the plantar reflex pass through the pyramidal tract, the cerebral centre is put out of action. But the spinal centre and its efferent and afferent paths remain intact, certainly for the toe-extension reflex, and if a spinal centre for toe-flexion exists, for this centre also. The activity of the spinal toe-flexion centre, which is a phylogenetically late reflex, need scarcely be considered as compared with the significance of the centre for the extension of the hallux, which is a very early reflex in phylogenesis. And though even the activity of this last centre is relatively small as compared with its own cerebral component, it is readily redeveloped. Consequently toe-extension reappears, though not in a very pronounced form. As at the same time the secondary tonus reflex is greatly increased owing to the enhanced irritability of its spinal centre, which is no longer controlled by the higher tonus-centres, we finally get an ample dorsal extension of the toe.

SUMMARY.

In this lecture I have tried to demonstrate the advantage to be derived from the use of the string galvanometer in clinical neurological research, where it can differentiate the several modes of muscle contraction. Graphic records of the electrical and mechanical responses in human muscles show both the innervation and the mode of contraction to be a double one. We have to distinguish between a tetanic and a tonic innervation as well as between contractions of tetanic and tonic character.

The graphic study of reflexes proves them to be much more complicated than is generally understood to be the case. The complication arises from the fact that every simple or tetanic reflex is in itself the cause for a secondary tonic reflex. Moreover most reflexes are governed by more than one centre. Application of these principles has been made to explain the genesis of the Babinski form of the plantar reflex.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

President—Dr. Ernest S. Reynolds.

In the Chair—Dr. W. Aldren Turner, C.B.

Meeting held October 7, 1920.

Aphasia: An Historical Review.

(THE HUGHLINGS JACKSON LECTURE FOR 1920.)

By Henry Head, M.D., F.R.S.

The evolution of our knowledge of cerebral localization is one of the most astonishing stories in the history of medicine. Throughout the Middle Ages the brain was supposed to contain three ventricles, each of which was the dwelling place of one or more aspects of the psyche [19]. The anterior chamber received the nerves of taste, smell, sight and hearing, and was the seat of the "sensus communis"; in the middle ventricle dwelt the faculty of cogitation and reasoning, whilst the posterior one was the seat of memory.

Under the profound influence of Descartes and his followers the vivid intellectual life of the seventeenth and eighteenth centuries swept away all traces of this scholastic materialism. Many guesses were made, but no satisfactory theory was evolved to account for the relation of mind and body; even as late as 1798, Soemmering thought the seat of the soul was in the fluid which filled the ventricles. No one presumed to imagine that the activities of the mind bore any direct relation to the life of the brain. Gall first suggested that this apparently uniform mass was made up of organs which subserved the manifestations of the vital and moral faculties of man. These could be divided into three groups: (1) Those concerned with the exercise of "vital force," which he located in the upper part of the spinal cord and medulla oblongata; (2) the "inclinations and affections of the soul," situated in the basal portions of the brain, and (3) the "intellectual qualities of the mind," associated with the cerebral hemispheres.

We habitually think of Gall with derision, as a quack who was responsible for phrenology and the study of bumps on the head. But it is to this man that we are really indebted for the beginning of the conceptions we now hold of the relations of the various portions of the nervous system to one another.

Franz Joseph Gall was born on March 9, 1758, at Tiefenbronn, in Baden. He studied medicine in Strasbourg, and in 1781 settled in Vienna. From 1796 to the end of 1801 he lectured on a new theory of the functions of the brain. His views were considered so subversive of religion and morals, that on December 24, 1801, his lectures were forbidden by an autograph letter of the Kaiser. Gall seems to have remained in Vienna for the next three years, and then journeyed through Europe with his pupil Spurzheim, lecturing as he went [10]. In 1807 he reached Paris, and in the following year presented a memoir to the Institute of France, entitled "Introduction au Cours de Physiologie du Cerveau." On this treatise a committee specially appointed drew up an unfavourable report. Gall and Spurzheim in 1809 published their "Recherches sur le système nerveux en général et sur celui du cerveau en particulier," which comprised a categorical examination and reply to the objections of the committee's report [11]. This was their first serious publication in French, and it contains all the views put forward by Gall at one time or another. Subsequently he issued in Paris a full account of his work in eight volumes, with an atlas of a hundred plates [12].

The doctrines of Gall had an enormous influence upon the thought of his time; but the only part of his theory which we attribute to him in the present day is the idea that the underlying structure of the brain has an effect upon the formation of the skull, and in consequence that character could be foretold from the external conformation of the head. But in reality this is but a small part of the theory put forward by Gall. He was the first to insist that the brain should be examined anatomically from the spinal cord upwards; for he laid down that the functions of the brain could only be explained by considering their relations to those of the spinal cord. All nerve fibres ended in grey matter, and the white matter of the nervous system served for conduction only. He pointed out the analogy between the grey matter which covers the hemispheres of the brain and of the cerebellum, with that which forms the corpora quadrigemina and optic thalamus. All nerves, whether they belonged to the brain or to the spinal cord, took their origin in grey matter, and the substance of the cerebrum and cerebellum could not be considered to be in direct connection with the peripheral nerves.

These ideas have been accepted into the body of general knowledge, but they were accompanied in Gall's exposition by the most fantastic theories with regard to the "moral" qualities. He pointed out that injuries to the head showed that the mind was associated with the brain, and that the brain serves as the organ of intellectual life. The nervous system of man was therefore the physical instrument of his moral life, for the nerves were the conductors by which the will was transmitted to all parts of the body. Then he set about to consider in what parts of the brain were situated the various moral and intellectual qualities of man. At that time it was supposed that man was endowed with certain "faculties," and it was these that Gall attempted to localize in different parts of the brain. He found the organ of the generative force in the cerebellum, and those of the five senses over the front portion of the brain. There were six different forms of memory, each of which he localized in a different "organ," or as we should say "centre." There was the memory of things, the memory of locality, the name-memory, the verbal and grammatical memory, and the memory for numbers. All of these were situated in those portions of the brain which lay in relation with the posterior part of the orbital cavity. It would seem that this localization dated back to Gall's earliest years; for as a boy he noticed that his companions who had "des yeux pochetés" (prominent eyes) had a gift for languages and a memory for words. He therefore concluded that the parts of the brain which pushed the eyes forward must be associated with the different varieties of verbal memory. Translated into anatomical terms, this meant that the frontal lobes and particularly their under surface must be devoted to these forms of memory.

He appears to have taken no precautions to verify this localization, but he had the opportunity of seeing an officer in whom a foil had passed through the base of the frontal lobes, producing grave disorders of speech. He gives a very short description of this case, but seems to look upon it simply as confirmatory evidence of his theory.

Gall died on August 22, 1828, and gradually most of his views passed into oblivion or came to be treated with derision; but his conception that speech and the memory for words were situated in the frontal lobes still found supporters. Its warmest champion was

Bouillaud, who was appointed Professor of Clinical Medicine at the "Charité" in 1831, and at the time that Broca made his first post-mortem was 65 years old, and the "doyen" of the faculty. He was an ardent follower of Gall, and offered a sum of money to anyone who would produce the brain of an individual who had lost his speech in which the anterior lobes presented no lesion.

In 1861 Broca had just become the head of the Surgical Unit at Bicêtre. An elderly man was admitted to his wards (aged 51) with a diffuse gangrenous inflammation of the right lower extremity. He was completely aphasic, and died a few days later; Broca demonstrated the brain at the Société d'Anthropologie, of which he was the secretary [5]. This brain showed destruction of the posterior part of the second and third frontal convolution, the inferior portion of the Rolandic area and the first temporo-sphenoidal convolution. It was preserved in the Musée Dupuytren, and was subsequently examined more carefully by Pierre Marie [16, No. 3], who has pointed out that the lesion also occupied the supramarginal gyrus, a part which at that time was not considered of any importance from the point of view of speech. This was followed by a second case [7] in which the situation and nature of the lesion was acknowledged to be more doubtful (November, 1861). This brain also has been examined by Pierre Marie, who could find no lesion of the third frontal convolution; but there seems to have been some wasting at the foot of the second frontal convolution, which is not uncommon in a senile brain. Broca himself says that this lesion is incomparably more circumscribed than that of the first brain, but he continues, "In comparing the two specimens it is possible to make certain that the centre of the lesion is identically the same in the two cases."

As may easily be imagined, these communications led to the greatest excitement in the medical world of Paris. Bouillaud, and his son-in-law Auburtin, greeted Broca as a convert to the doctrines of Gall. The localization of speech became a political question; the older conservative school clung to the conception that the brain "acted as a whole," whereas the younger liberals and republicans passionately favoured the localization of function. For the next few years, every scientist of importance in Paris took one side or the other. Trousseau brought the weight of his authority into the scale on the side of Broca, and invented the word "aphasia" to replace Broca's "aphemia." This was an unjustifiable change in nomenclature, which as Broca complained "brought back into pathology the very confusion I thought to dissipate."

Broca described aphemia as follows: "There are cases where the general faculty of language persists without alteration, where the auditory apparatus is intact, where all the muscles, without excepting those of the voice and articulation, obey the will, and where in spite of this a cerebral lesion abolishes articulate language; this abolition of speech in individuals who are not paralysed or idiots constitutes a symptom sufficiently singular to be given a special name. I shall give it the name of 'aphemia' because what is wanting in these patients is solely the faculty of articulation. They hear, they understand all that one says to them; they have their intelligence, they can emit vocal sounds with ease; they can execute with the tongue and cheeks movements much more energetic than those required for the articulation of sounds. Moreover, the answers they can make are reduced to a very few articulated sounds, always the same and always disposed in the same manner. Their vocabulary is composed of a short series of syllables, sometimes of a monosyllable which expresses everything, or rather it does not express anything, because the single word is often foreign to all vocabularies. Some patients have not even this vestige of articulated language; they make vain efforts without pronouncing a syllable" [6].

Broca had discovered a definite clinical condition, which he called "aphemia," produced by destruction of the substance of the brain, and he believed that he had found the seat of the faculty of articulated language in the third frontal convolution.

Interest in the question of localization of speech rapidly spread to England, and in 1864 Hughlings Jackson published his first paper on "Loss of Speech with Hemiplegia of the Left Side" [14, No. 2]. This was followed by a series of contributions on the subject in which many other physicians joined.

In 1868, the British Association for the Advancement of Science held its Annual Meeting at Norwich, and M. Broca opened a discussion on aphasia. He was followed by Dr. Jackson. No record of these contributions is to be found in the *Proceedings of the British Association*, but Broca's paper was published in the *Tribune Médical* [8].[1] An abstract of Jackson's paper appeared in the *Medical Times and Gazette* for September 5, 1868. This has been reprinted amongst the collected papers in *Brain* [14, No. 17].

---

[1] I was unable to find this paper in London and owe my knowledge of its contents to the kindness of Professor Pierre Marie, who caused a copy to be made by hand. This he presented to me in memory of his master, Broca, for whom he acted as Interne in 1880.

Thus, we are able to compare the views of the French and English protagonists of localization, and it is no wonder that at that period, and, under those conditions, Jackson's views produced little serious effect. Broca is clear, definite and precise, and I shall therefore begin this stage of the history by summarizing his views.

He divides affections of speech of central origin into two main groups —"Aphémie" and "Amnésie verbale," and lays down clearly the difference between them. The aphemic patient has a profoundly reduced vocabulary and may be speechless, except for some monosyllables, oaths and words that do not seem to belong to any language. His ideas are intact, as shown by gestures, and he can understand what is said to him and recognizes words and phrases which he cannot pronounce or even repeat. On the other hand, the amnesic patient no longer recognizes the conventional associations established between ideas and words. He can pronounce them, but they do not seem to have any bearing on the ideas he wishes to express. He is able to show by gestures that he has not lost all kinds of memory, but it is the special memory, not only of spoken but of written words, that is lost.

So far Broca's statements are admirably clear and consonant with fact. When however he tries to determine the site of the lesions responsible for these conditions, he becomes confused and indefinite. He saw clearly that the larger number of cases did not correspond to these two clean-cut divisions. In practice, both states can exist together and this greatly hampered the determination of the position of the lesion. He utters a wise warning on the necessity for drawing conclusions from selected cases only, and insists that it is "aphemia" which is caused by a lesion of the third frontal convolution.

Let us now turn to Hughlings Jackson's contribution to this discussion. He said nothing about localization beyond referring to his previous papers. As far as can be gathered from the summary [14, p. 59], he confined his attention to the nature of the loss of speech produced by cerebral lesions. He pointed out that healthy language consisted of two inseparable, yet distinct, forms: (1) Intellectual—i.e., the power to convey propositions; (2) emotional—i.e., the ability to exhibit states of feeling. These two are separated by disease. It is intellectual language alone which suffers in most of these cases; emotional language usually escapes altogether. Cases of affection of speech can be divided into two groups, in the first of which the patient is speechless, or nearly so; whilst in the second, he has plenty of words but uses them wrongly. He denied that there was such a thing as a "faculty of speech" or a "faculty of memory," and says "The question is not, how is general mind damaged, but what aspect of mind is damaged?"

Thus, he laid down in this discussion the fundamental basis of the whole of his future treatment of the subject. It is the power to form propositions that is affected, and not the memory of words or faculty of language. The higher the propositional value of the task the patient is asked to carry out, the less he will be able to respond; whereas, the lower or more automatic the action required, the easier will it be to perform.

But subsequent investigators took no care to emulate the clinical acumen of Broca or the psychological insight of Hughlings Jackson. Bastian published his well-known paper in 1869 which had such an evil influence on the subsequent course of the discussion. He started from the *a priori* assumption that we think in words, and that words are revived in the cerebral hemispheres as remembered sounds. He talked of lesions of special fibres and centres, and set the points on the catastrophic road to schemas and diagrams. His subsequent Lettsomian Lectures [3], re-published in 1898 under the title "Aphasia and other Speech Defects," had, as a frontispiece, the famous diagram so often cited in subsequent discussions [4]. This book is little more than a widely expanded version of his original paper, and by its simplicity and dogmatism seduced the younger generation away from the difficulties and complexities of Jackson's doctrine.

The first actual diagram was published by Baginsky in 1871 [1], who like most of the Germans was unaware that Bastian had already described word deafness. From this time onwards the rage for diagrams became a veritable mania. No one could write on aphasia without producing a new diagram of centres and the paths between them. Each author twisted the clinical facts to suit the lesions he had deduced from his pet schema. Wernicke in 1874 [21] localized the centre for auditory images in the first left temporal convolution, but would allow no centre for reading and writing; oral speech alone possessed cortical centres, one of which was guiding and the other emissive. Between these two lay the island of Reil, and three main forms of aphasia must therefore be possible. The first of these was sensory, and the lesion was situated in the temporal convolution. The second was motor and the lesion lay in the third frontal convolution. The third variety was due to interference with conduction between these two centres.

How far the writers of this period were compelled to lop and twist the clinical facts to fit the procrustian bed of their hypothetical conceptions is shown by the famous case published by Wernicke in 1903 as "A Case of Isolated Agraphia" [22]. Now, such a title can only mean that in this patient every other act of language could be perfectly performed, except that of writing. But when the record of the case is examined, the patient appears to have had much difficulty with spontaneous speech, and in understanding what was read. She could not comprehend many spoken words, nor could she carry out commands given verbally. She is said to have shown almost complete inability to write, and it was with great difficulty she could be brought to make the attempt. She was also unable to draw to command, although she could copy drawings and writing, and she failed to say the days of the week or the months in their proper order. In the solemn discussion which follows, on the nature of disorders of speech, it is difficult to decide whether the clinical obtuseness or want of theoretical insight is more worthy of wonder.

Amongst the various diagrams that of Lichtheim [15] attained the greatest popularity. It was definite and precise; every form of aphasia was accounted for by a lesion of some hypothetical centre or purely imaginary path. Teachers of medicine could assume an easy dogmatism at the bedside and candidates for examination rejoiced in so perfect a clue to all their difficulties. But serious students could not fit these conceptions of aphasia with the clinical manifestations; incredulous of such scholastic interpretations, they lost interest in a problem of so little practical importance.

In 1906, Pierre Marie startled the medical world by three papers in the *Semaine Médicale* on "Revision of the Question of Aphasia" [16]. The first of these bore the aggressive title, "The third left frontal convolution does not play any special rôle in the function of language," whilst the third was a fascinating historical essay on "Broca and his times," together with an account of the re-examination of the hemispheres of his two first patients. This was followed by a series of papers on the subject of aphasia and its cerebral localization, culminating in the monograph of Moutier which appeared in 1908 from the laboratory of Pierre Marie at Bicêtre [17].

Marie laid down that the aphasia of Broca was not a clinical entity but a "syndrome." It is a combination of two distinct troubles which he called "anarthria" and "aphasia." By "anarthria" he understood simply the inability or difficulty of articulating words. On the other hand, aphasia was the sensory aphasia of Wernicke, characterized by troubles of internal language, which showed themselves directly in alterations of speech, reading and writing. There is nothing of aphasia in the motor trouble of the anarthric. He understands, he reads and he writes. His power of thinking is intact, and expression is possible by every other means except words, internal language remaining unaltered. There is only one aphasia, the aphasia of Wernicke, and the term "sensory aphasia" must disappear.

But the greatest excitement was produced by Marie's views on the cerebral localization of the lesion responsible for these defects of language. Of the two factors which constituted the aphasia of Broca, the anarthric aspect was due to a lesion of the "lenticular zone" whilst the aphasic side was produced by one in "Wernicke's zone." This latter was not a special loss of word memories, but was a defect of general intelligence, and of special intelligence of language.

In Moutier's monograph, "L'Aphasie de Broca," he gives an interesting history of our knowledge of aphasia, followed by a reconstruction of the nature and symptoms of this disorder from the point of view of his master, Pierre Marie. One of the most valuable portions of this book consists in a series of reports, both clinical and pathological, of a large number of cases. The second part is prefaced by a scheme of examination which is singularly complete.

By this time the differences of opinion in Paris had become so acute, that three sittings of the Société de Neurologie de Paris (June 11, July 9, and July 23, 1908) were devoted to a discussion of this question [9]. Unfortunately, though an attempt was made to keep the clinical and pathological aspects apart, this was impossible in practice. Two questionnaires were prepared to serve as the basis for discussion. The first of these was divided into "Étude anatomique" and "Étude clinique," whilst questionnaire B consisted of three portions: (1) "Clinique"; (2) "Anatomie normale et pathologique"; (3) "Physiologie pathologique." The second of these was accepted as the basis of discussion, which began as follows: "Are motor and sensory aphasia clinically different from one another, or is motor aphasia sensory aphasia accompanied by anarthria? If there are differences, what are they?" Unfortunately this led to a discussion based entirely on terms such as "Broca's aphasia," "total aphasia," "word blindness," "word deafness," "alexia," "agraphia," &c. Marie held strictly to the views set out above, whilst Dejerine defined his position as follows: "The aphasia of Broca is a motor aphasia and total aphasia is both sensory and motor. The aphasia of Broca is accompanied by a loss of words, marked trouble in writing, some alexia, though not excessive; the patient is able to transcribe print into handwriting. On the other hand the sensory element consists in word deafness and word blindness.

Dejerine attacked the use of the word "anarthria," which should, he contended, be kept for definite troubles of articulation Marie represented that he had chosen this word because cases of mere motor aphasia showed no disturbance of internal speech.

Dupré and others insisted that the motor aphasic had difficulty in evoking his words. Aphasia is a psychical defect and trouble of language ("langage"); anarthria, on the other hand, is a motor defect and a trouble of speech ("parole"). Marie answered that the anarthric had no trouble in evoking a word, but solely in pronouncing it, and he agreed to substitute the word aphemia for anarthria, provided the meeting would acknowledge that the phenomenon was not associated with a defect of internal speech.

The next sitting and the first half of the final sitting were devoted to an attempt to settle the situation of the lesion responsible for the various forms of aphasia. But, as no satisfactory clinical definition had been arrived at during the earlier phase of the discussion, the greater part of the time was spent in discussing the limits of Marie's lenticular zone and its justification. Finally, the meeting turned to a consideration as to whether there were disturbances of intelligence in motor aphasia, and if so, what form they took, and secondly, whether intelligence was disturbed in sensory aphasia. At this stage, Marie brought forward his views on the evident diminution of general intellectual capacity, which is found in those cases which he describes as the "aphasia of Broca." This is in no sense a dementia; he insisted on the exactness of the terms that he had used. "Il y a une diminution très marquée dans la capacité intellectuelle en générale" (p. 1037). This intellectual disturbance is a specialized defect. He admitted that the "zone of Wernicke" is the part where a lesion produced with certainty troubles of language, but he denied independent existence of special sensory word centres which had been attributed to this portion of the brain.

Marie fought hard for simplification and made a bold attempt to sweep away the vast accretion which had obscured the question of aphasia; but no one ventured to suggest that the greater part of the discussion of the clinical manifestations was purely a verbal battle. Had the disputants been familiar with the work of Hughlings Jackson, they would have recognized that, from the point of view of the question at issue, all these terms such as "aphasia of Broca" "total aphasia," "word blindness," and "word deafness," were pure phrases, and did not in reality correspond exactly to any clinical phenomena.

I have kept the views of Hughlings Jackson until the last, because his attitude towards the question was entirely foreign to any conception put forward in the history of aphasia. His views bore no fruit during his lifetime for two reasons. Firstly, they were published in a series of papers between 1864 and 1893, many of which were inaccessible on the Continent. Secondly, the style in which they were written makes them peculiarly difficult to read. He was so anxious not to overstate his case that almost every page is peppered with explanatory phrases and footnotes, so that the generalization can scarcely be distinguished from its qualifications. English students, accustomed to the fluent dogmatism of his contemporaries, turned away from the bristling difficulties of Hughlings Jackson's papers. Finally, the ideas he propounded were entirely opposed in their nature to the current opinions of the day. No one assimilated his views on defects of function and applied them to actual cases of loss of speech. We failed to appreciate how much closer these conceptions would have led us to the phenomena of aphasia than the glib generalities founded on the supposed anatomical facts of cortical localization.

When however I became interested in working out the problem of aphasia in the light of our researches on the effects produced by cortical lesions on sensation, it became obvious that none of the current doctrines corresponded to the clinical facts. I set to work to make a collection of Jackson's papers on the subject, and was startled to find that he had long ago arrived at several of the general conclusions which had led me to disbelieve the teaching of my contemporaries. In 1914, Pick's remarkable book, "Die agrammatischen Sprachstörungen" [18] fell into my hands and determined me to republish Jackson's papers on aphasia in *Brain* [14].

In spite of its occasional obscurity Jackson's work is of peculiar importance to the neurologist of to-day, both as a practical guide to the clinical phenomena of aphasia and an understanding of the processes which underlie the production of speech. His views can be summed up shortly as follows:—

(1) In 1868, he pointed out that patients with aphasia can be divided into two main groups. In Class 1 the patient was almost speechless or speech was gravely damaged, but in the worst of these cases the patient

could utter some one or two unvarying words or jargon. Class 2 comprises those who have plentiful words but habitually use them wrongly.

(2) The loss of power to carry out an order depends on the complexity of the task. The more abstract the conception, the more likely is the patient to fail in executing it, although he may succeed when it is put before him in a simpler and more descriptive form. Thus, the patient who could not find the word "kitten" called it "a little fur child," and one who could not draw a square when asked to do so, drew a perfect square when told to draw a block of wood. Thus, it is most important, when asking an aphasic patient to carry out some order, to present it to him if possible in several forms, noting his response in each case; because a man cannot write the alphabet, we must not assume that he cannot write a letter.

(3) The higher and more voluntary aspects of speech tend to suffer more than the lower or automatic. The least voluntary speech is that which is emitted under the effect of emotion, such as exclamations, oaths, and words such as "good-bye," rising to utterance under the impulse of a moment. In many cases "yes" and "no" can be used approximately as propositions, and even words and phrases of true propositional value may spring to the lips of the aphasic patient. But in such cases he is usually unable to repeat at will the phrase he has just used under an appropriate impulse. The "speechless man is not wordless," and the apparent inconsequence of observations on persons with aphasia is to a great extent removed by an analysis of the conceptual value of the words and phrases which are actually uttered.

(4) Writing is affected, not as a separate "faculty" but as a part of the failure to propositionize in words. There is no such condition as pure "agraphia." A man who cannot write spontaneously may be able to copy printed matter in perfect handwriting. The "faculty" of relating handwriting to print is intact; he cannot write voluntarily because he has lost the use of written words in propositions. Hence, he can usually write his name and address, because in most of us this has reached with time more nearly the level of an automatic act.

(5) Patients with such affections of speech may not be able to read aloud or to themselves, when asked to do so; but they can understand what is read to them and may even obey written commands, although unable to reproduce them in words. This is not due to some loss of function called "alexia," but to an inability to reproduce a proposition which, on the other hand, they may be capable of receiving accurately.

(6) "Imperception" is on the receptive side what aphasia is in the

"word series." In many cases the two conditions are combined; but they may exist separately, and, where aphasia is present without "imperception," images remain intact. Thus the patient may be able to point to colours and objects when they are named; he continues able to play cards or dominoes; he recognizes handwriting, although he cannot read the words written; he knows poetry from prose by the different endings of the lines on the right of the page.

Thus, affections of speech are caused (*a*) on the emissive side by inability to form or to express a proposition in words; (*b*) on the receptive side by failure of those mental processes which underlie perceptual recognition.

(7) External and internal speech are identical, except that one leads to the utterance of articulated words, whilst the condition of internal speech can be discovered by writing only.

(8) Behind external and internal speech stands the proposition which, when verbalized, can be expressed in speech or writing. This proposition is necessary for clear and logical thought, but not for all thinking. When this aspect of speech is affected, the patient cannot retain a sequence of abstract propositions, because he is unable to formulate them at will to himself. He can think, but he is "lame in thinking."

(9) If, however, "imperception" is added to such defect of speech the patient will not only suffer from "inferior speech," but will show signs of "inferior comprehension."

(10) In the majority of cases of affections of speech mental images are unaffected. This extremely important contribution to the theory of aphasia has been entirely neglected by neurologists. For almost every hypothesis propounded in the last forty years presupposes some defect in "auditory" or "visual word images."

In 1910, it became obvious to me in the light of our work on the part played by the cortex in sensation, that new tests must be devised before the clinical investigation of aphasia could produce any satisfactory results. It is not sufficient to hold up some object and ask the patient to name it; at one time he may be able to do so, at another he fails completely. No conclusion can be drawn from one or two questions put in this way. His power of responding must be tested by a series of observations in which the same task occurs on two or more occasions.

Not only is it necessary to arrange the tests in sequence, but each set must be put before the patient in several different ways. For example, six common objects are laid on the table in front of him, and he is asked to point to the one which corresponds to a duplicate placed in his hand out of sight. This is repeated for from eighteen to twenty-four observations, so that the choice of any one object recurs three or four times in the course of the series. Then he indicates each one in turn as it is named by the examiner, or makes his selection in answer to printed words set before him on a card. He next gives names to the various objects, one by one, and finally writes them down without saying anything aloud. The order in which these various acts are performed remains the same throughout the different aspects of the test. This alone makes it possible to draw any conclusion from the inconstant responses, which are so disconcerting, unless the answers are recorded in this manner. Moreover, this method enables us to learn how the patient responds to the same series of tests put before him in different ways. I have described all these methods of examination in a recent number of *Brain*, and shall not dwell further upon them [13]. The principle upon which they are based is to set a similar task before the patient through various forms of command; these he must execute by different methods. He is no longer asked to speak, to read or to write; but all these methods of expression are used to answer the same series of tests.

As soon as I began to apply these methods of examination to patients suffering from aphasia, it became obvious that none of the ordinary theories bore any relation to the facts I discovered; it was not until I had read through a complete series of Jackson's papers that I recognized the importance of the principles he had enunciated in the light of my own observations.

Shortly after the beginning of the War, patients began to pass into my care with wounds of the head, who suffered from defects of speech slighter and more specialized than any I had seen before. Moreover, these young men were struck down in the full pride of health; many of them were extremely intelligent and anxious to be examined thoroughly. As their wounds healed, they were encouraged and cheered by the obvious improvement in their condition. They were euphoric rather than depressed, and in every way contrasted profoundly with the state of the aphasic patient met with in civilian practice. From this time onwards the work progressed rapidly, and the results that I shall lay before you are based mainly upon these cases of unilateral wounds of the head.

But, before we enter on the positive results of these researches, I am anxious to clear the ground of certain preconceptions. We know that speech can be affected by destruction of the substance of the brain, but this does not show that "the faculty of speech" is localized in any area of the cortex. We should as soon expect a special centre for eating as for speech; both are complex acts which do not correspond to any specific group of functions. No lesion, however local, can affect speech and speech only. The cerebral injury disturbs certain physiological processes which subserve the complex acts which we speak of as speech. Now, no anatomical lesion corresponds exactly to a single group of physiological functions; the acuteness and severity of the onset is often a more important factor in determining their nature than the extent of the lesion. On the other hand, there is no single psychological function or "faculty" corresponding to speech; any organic injury which produces a disorder of speech disturbs other functions not usually associated with language, or leaves unaffected much that undoubtedly belongs to speech. We must therefore get rid of all those *a priori* conceptions, which underlie such terms as "motor" and "sensory" aphasia, "alexia," "agraphia," or "amnesia verbalis." No disorder of speech due to a unilateral lesion of the brain corresponds exclusively to any one of these categories, still less can these hypothetical conditions be associated with limited destruction of any one part of the brain.

An organic lesion disturbs certain physiological processes which are necessary for the complex acts which underlie the use of language. Words, numbers, pictures, and every function which depends upon the use of these symbols in constructive thought may be affected. Any mental process is liable to suffer which demands for its performance exact comprehension, voluntary recall, and perfect expression of symbolic representations. I have therefore suggested that the various functions disturbed in aphasia and allied conditions might be spoken of as "symbolic thinking and expression," because it is mainly the use of words, numbers and pictures which suffers in these disordered states. But this name must not be taken as in any way defining the group of processes affected; I should have preferred, had it been possible, to have employed some entirely indifferent term. For it is symbols used in a particular manner which suffer in these disorders and not all symbolic representations.

The more nearly the symbolic action approximates to a perfect proposition, the greater difficulty will it present, and the patient will probably fail to execute it correctly. Conversely, the more closely it corresponds to matching two sensory patterns, the less likely is it to be disturbed. Highly complex symbolic acts suffer more gravely than those of lower propositional value. For example, if a patient seated opposite the observer attempts to imitate a series of movements which consist in touching the eye or ear with one or other hand, he may fail grossly. He can however execute them perfectly when the observer stands behind him and they are reflected in a mirror. For in order to imitate movements sitting face to face he must formulate the various factors which make up the command; whilst to copy them when reflected in a mirror is an act of almost pure imitation. But if, instead of imitating the movements, he is asked to write down what he sees in the mirror, he again fails grossly; for he is now compelled to formulate the task in words, and so fails to describe gestures he can imitate perfectly.

The higher the propositional value of the mental act, the greater difficulty will it present. Thus, the patient may be able to execute a printed command to hold up his hand, although he is unable to carry out an order to touch with it his eye or his ear. The addition of the second factor has rendered the task too difficult. The larger the number of alternatives presented by the order, the more certainly will the desired action be defective. All propositions express an abstract relation; but even the same generalized statement may vary in difficulty according to the means by which it is expressed. For example, during the compass test, the aphasic patient may be entirely unable to record in speech, or in writing, whether he has been touched with one or two points; but if the figures 1 and 2 are written on a sheet of paper, he can indicate correctly whether the contact was single or double. Under the ordinary conditions of this test, he is not only compelled to formulate in words his sensory impressions, but he must express them in verbal symbols; but when it is carried out according to the second method, he has only to determine whether his sensations are of one or two points, and the means of expression lie on the table before him. When the defect consists of want of power to evoke words or names, the patient may be unable to name a set of common objects, although he can choose them correctly to oral or printed command. The verbal symbol conveys its proper meaning when presented ready formed in sound or in print; but to produce it freely at will demands a greater perfection of symbolic thinking and expression. In the same way when an aphasic calls scissors "that is the tweezers to cut with," he is expressing a lower grade of symbolic recognition. One of my patients was unable to name a series of colours placed before him, and could not choose them correctly to oral or printed commands. But if instead of the names he was allowed to call black, "what you do for the dead"; red, "what the staff wear," with similar descriptive phrases for each of the other colours, he named them all correctly. These metaphorical modes of expression are simpler than names which consist of a single word; for the more concrete is lower in the hierarchy of mental processes than the general and abstract.

Behind every propositional expression lies recognition of the meaning of symbols; I must be able to appreciate the full significance of words, numbers, and pictures before I can pose them in propositional form. This loss of meaning is the cause of many aphasic defects; thus, the short and the long hands of the clock have acquired a significance which converts each one of them into a direct symbol, and they are often confused, or used wrongly in cases of aphasia.

When adding two numbers together, it is easier to reach the answer by counting than by direct proposition. Thus, many aphasics can add 6 and 3 by saying to themselves 6, 7, 8, 9, but cannot formulate 6 + 3 = 9. For the same reason some of them could set the clock correctly to a command given in railway time, but not if it was expressed in words. Told to set the clock at 4.45, the patient placed the hour hand at 4, and then swept the other round the face to a position he associated with 45 minutes. But asked to set a quarter to five, he placed the one hand at 5, whilst the other hovered uncertainly between a quarter past and a quarter to. This does not depend on a difference in the power of comprehending words and numbers; for when the patient attempted to tell the time it was equally difficult for him to evoke the names "4.45" and "a quarter to five."

Many of the disorders found in aphasic patients are the result of inability to recognize the significance of words and names. If, however, they fail to appreciate with certainty the ultimate meaning in thought, they are liable to become confused over the intention of an action, whether self-suggested or imposed from without. The patient no longer perceives clearly the goal at which he must aim. Thus, a young officer could thread a quadrilateral frame for his bee-hives if the action consisted in bringing the wire across from one side to the other and back again through neighbouring holes, but as soon as he attempted to go from corner to corner, he failed entirely.

Perfect symbolic thinking and expression demand that words, numbers, pictures, and all they stand for in thought should be susceptible of voluntary manipulation. It is this aspect that is usually affected in the more verbal forms of aphasia and in those that are characterized by the use of jargon; these are not due to "dysarthria," but are produced by defective conceptions of the structure and rhythmic balance of the symbol, which interferes not only with articulated speech but also with internal verbalization. All these functions are not uniformly disturbed in every instance, and aphasics differ profoundly in their clinical manifestations. The majority, especially in the earlier stages, show evidence of widespread defects in the use of language; the more acute and severe the lesion, the graver and more extensive will be the disorder it produces. Many of these changes may disappear, and sometimes the patient recovers entirely; but, in serious cases, one or more aspects of symbolic thinking and expression remain affected over a considerable period, and the aphasic manifestations consequently assume some particular form.

Analysis of these clinical varieties seems to show that the various functions included under the term "symbolic thinking and expression" can be dissociated in different ways under the influence of organic injury. They do not correspond to different stages of dissolution or recovery; nor do they reveal directly the elements out of which language is built up. On the contrary, they show the components into which a highly complex set of psychical processes can be separated by destruction of certain portions of the brain. Provided we bear these principles in mind, we are justified in grouping the clinical manifestations under separate headings. Each case of aphasia is the result of the loss of one or more of these groups of functions; fortunately, all of them are rarely affected in the same patient, for, if this were the case, he would be reduced to a state in which all detailed examination would be impossible. Thus, in each patient we must determine which functions are disturbed and which have remained unaffected. By careful analysis it seems possible to separate these disorders of speech into four main varieties; but it must be remembered that they can appear together in every combination.

To each of the groups I have given the name chosen from the most salient defect in the use of words.

(1) *Verbal aphasia.*—This is essentially a disturbance of word formation; words are evoked with difficulty, the patient's vocabulary is greatly restricted, and enunciation is slow and halting. Writing tends to show the same sort of errors as articulatory speech and spelling is defective. The patient has difficulty in reading to himself with pleasure, because he is unable to retain in his memory a long series of words.

Numerals are affected to a slighter degree; their significance may be recognized and acted on correctly, although they are wrongly enunciated. As speech returns, commands given in spoken or printed words can be executed, but orders which necessitate the evocation of some word or phrase may be carried out badly. These patients recognize, however, whether the task they are attempting has been performed correctly or not. They can draw, play card games, and appreciate jokes set out in print or in pictures. It is this group of defects which is mainly responsible for the condition described by Broca as "aphemia" and usually spoken of as slight "motor aphasia."

(2) *Syntactical aphasia.*—This is an easy form to distinguish because the patient tends to talk jargon; not only is articulation of the word ill-balanced but the rhythm of the phrase is defective, and there is want of grammatical coherence. Speech, once started, is voluble and words are emitted with great rapidity; sometimes each one is comprehensible, however difficult it may be to gather the full meaning of the phrase; but in other cases the words uttered are pure jargon. The power of naming objects placed before him may be retained by the patient, in spite of the jargon by which he is hampered. Not infrequently, when he cannot utter some word, or when the sound emitted is incomprehensible to his auditor, he can write the name correctly. These patients can read if they are not compelled to reproduce the meaning in words; writing is usually less affected than external speech, and single words, especially names in common use, can be written correctly; but any attempt to convey a formulated statement is liable to end in confusion. This form was described by Hughlings Jackson, but has not received the attention it deserves except in the writings of Pick.

(3) *Nominal aphasia.*—This is essentially a defective use of names and want of comprehension of the nominal meaning of words or other symbols. The patient reads with extreme difficulty, especially if he attempts to spell out the words; writing is gravely affected, but he may be able to copy print into cursive handwriting. Writing to dictation and all actions demanding choice are performed with difficulty to spoken command; counting is possible to a varying extent, but the significance of numbers, the power to carry out simple arithmetical operations, and appreciation of the relative value of money are usually more or less affected. The patient is unable in most cases to draw an accurate ground plan of some room with which he is familiar; he cannot play cards, but chess and draughts may be possible. This form cannot be fitted into any of the older methods of classification; for, on the one hand, it has

some of the characters of "motor aphasia," whilst, on the other, other defects would have been attributed to "sensory aphasia." The separation of word-formation from naming and its allied functions is an entirely new feature in the classification of the aphasias.

(4) *Semantic aphasia.*—So far, the names I have applied to the various forms of disorders of speech have borne some relation to the verbal defects; for this group, however, it is difficult to find a suitable designation that will express the essential nature of the disturbance which extends beyond the limits of organized words. I have chosen the term "semantic" as a label for this form of aphasia, because the affection comprises want of recognition of the full significance or intention of words and phrases. The patient may understand each word or short phrase exactly as he can comprehend the details of a picture, but the ultimate meaning escapes him. He fails to grasp the final aim or goal of the action imposed upon him from without; he cannot formulate symbolically a general conception, although he can enumerate the details of which it is composed. He can read and write, but the result tends to be inaccurate and confused; counting is possible and the value of numerals can be recognized, but arithmetical operations are impossible, or are carried out uncertainly and with difficulty. The patient is unable to add or subtract, because the mathematical process itself is incomprehensible. He fails entirely to comprehend most jokes, especially if they demand the complete understanding of a picture or its legend. He cannot play card games or put together puzzles, which confuse him greatly. These semantic disorders interfere seriously with the actions of daily life and render the patient useless for any but the simplest employment; and yet his memory and intelligence may remain on a comparatively high general level. He does not forget people or places, and can recall accurately events, both recent and remote; but he has lost the power of appreciating the ultimate or non-verbal meaning of words and phrases, and fails to recognize the intention or goal of actions imposed upon him.

I have attempted to put before you shortly a conception of the phenomena of aphasia, based on a systematic re-examination of the clinical facts by new methods. The results I have obtained are of no direct practical value to the physician, but they form a fascinating example of the interaction of body and mind—one, moreover, capable of experimental demonstration. This problem has occupied the attention of the acutest human intellects for over two thousand years. The schoolmen answered the eternal riddle in material terms founded on imaginary anatomy. They were followed by the philosophers, who, for the most part, spun their theories out of *a priori* conceptions. Then came the era of the physicians, who were unwillingly forced to face the question, because the most illuminating examples of the effect of bodily conditions on the state of the mind were entrusted to them for therapeutic supervision. They developed an extensive and accurate acquaintance with the anatomy of the brain, but employed the fruits of this great advance in knowledge in the service of an obsolete and almost mediæval psychology. Fifty years ago Hughlings Jackson raised the question of disorders of speech to a higher plane by rejecting all *a priori* conceptions; but he stood alone and medicine turned a deaf ear to his teaching. To-day, however, the older psychology is tottering to its grave, and I am proud to think that a long continued and arduous series of researches has led me at last to understand that great empiric philosopher, the founder of English neurology.

REFERENCES.

[1] Baginsky. "Aphasie in Folge schwerer Nierenerkrankungen," *Berl. klin. Wochenschr.*, 1871, S. 441.

[2] Bastian, H. Charlton. "On the Various Forms of Loss of Speech in Cerebral Disease," *Brit. and Foreign Med.-Chir. Rev.*, 1869, January-April, xliii, pp. 209-236, 470-492.

[3] *Ibid.* "On some Problems in connection with Aphasia and other Speech Defects," (the Lettsomian Lectures), *Lancet*, 1897, i, pp. 933, 1005, 1131, 1187.

[4] *Ibid.* "Aphasia and other Speech Defects," Lond., 1898.

[5] Broca, P "Perte de la parole. Ramollissement chronique et destruction partielle du lobe antérieur gauche du cerveau," *Bull. Soc. Anthropol.*, 1861, 18 avril, ii, pp. 235-238.

[6] *Ibid.* "Remarques sur la siège de la faculté du langage articulé, suivies d'une observation d'Aphémie," *Bull. Soc. Anat.*, 1861, vi, pp. 330-357.

[7] *Ibid.* "Nouvelle observation d'aphémie produite par une lésion de la troisième circonvolution frontale," *Bull. Soc. Anat.*, 1861, vi, pp. 398-407.

[8] *Ibid.* "Sur la siège de la faculté du langage articulé," *Trib. Méd.*, 1868, No. 74, pp. 254-256, and No. 75, pp. 263-269.

[9] "Discussion sur l'Aphasie," *Rev. Neurol.*, 1908, xvi: Séance du 11 juin, p. 611; Séance du 9 juillet, p. 974; Séance du 23 juillet, p. 1025.

[10] Gall, F. J. "Craniologie ou découvertes nouvelles du Docteur F. J. Gall." Ouvrage traduit de l'Allemand, Paris, 1807.

[11] Gall, F. J., and Spurzheim, G. "Recherches sur le Système Nerveux en général et sur celui du Cerveau en particulier," Paris, 1809.

[12] *Ibid.* "Anatomie et Physiologie du Système Nerveux en général et du Cerveau en particulier, avec des observations sur la possibilité de reconnoître plusieurs dispositions intellectuelles et morales de l'homme et des animaux par la Configuration de leur Têtes" (4 vols. in folio, 4 vols. in quarto, atlas of 100 plates), Paris, 1810-19.

[13] Head, Henry. "Aphasia and Kindred Disorders of Speech," *Brain*, 1920, xliii pp. 91-107.

[14] JACKSON, J. HUGHLINGS. "Papers on Aphasia and Kindred Affections of Speech," reprinted in *Brain*, 1915, xxxviii, pp. 1-190.

[15] LICHTHEIM, L. "On Aphasia," *Brain*, 1885, vii, p. 433.

[16] MARIE, PIERRE. "Revision de la Question de l'Aphasie," *Semaine méd.*, 1906. (1) "La 3e circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction du langage," 23 mai, pp. 241-247; (2) "Que faut-il penser des aphasies sous-corticales?" 17 octobre, pp. 493-500; (3) "L'aphasie de 1861 à 1866; essai de critique historique sur la genèse de la doctrine de Broca," 28 novembre, pp. 565-571.

[17] MOUTIER, F. "L'aphasie de Broca" (Travail du laboratoire de M. le Professeur Pierre Marie), Paris, 1908.

[18] PICK, ARNOLD. "Die agrammatischen Sprachstörungen: Studien zur psychologischen Grundlegung der Aphasielehre," 1 Theil (the second part has not appeared), Berl., 1913.

[19] SUDHOFF, W. "Zur Lehre v. d. Hirnventrikeln in textlicher v. graphischer Tradition d. Altertums u. Mittelalters," *Arch. f. ges. d. Med.*, 1913, vii, S. 149.

[20] VILLERS, C. "Lettre de Charles Villers à Georges Cuvier sur une nouvelle Théorie du Cerveau par le Docteur Gall; ce viscère étant considéré comme l'organe immédiat des facultés morales," Metz, an x (1802).

[21] WERNICKE, C. "Der aphasische Symptomencomplex," Breslau, 1874.

[22] *Ibid.* "Ein Fall von isolierter Agraphie," *Monatschr. f. Psych. u. Neur.*, 1903, xiii, S. 241.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

President—Dr. ERNEST S. REYNOLDS.

Meeting held November 11, 1920.

Discussion on Aphasia.

DR. HENRY HEAD, F.R.S., said that (1) it was universally acknowledged that speech could be affected by destruction of the substance of the brain. If the lesion was acute, all aspects of the act of speech might be generally reduced. As the nervous tissue recovered, all these functions might return more or less together, and the aphasia disappeared without the manifestations assuming at any time a specific form. This generalized loss of function was particularly liable to occur when the disorder of speech was produced by vascular lesions or by any condition that lowered the vital activity of the brain. In cases of head wounds it was therefore important to wait until the original nerve shock had passed away and the wound had healed, before attempting to draw any conclusions as to the supposed specific character of the loss of speech. It might happen, however, after the acute stage has passed away, that the lesion of the brain was found to have affected the production of those physiological activities which underlay one or more special aspects of the perfect use of language and allied functions. Speech consequently assumed in such cases a clinically specific form.

(2) The categories of such partial forms of aphasia could only be drawn up in terms of the tests employed; they did not correspond to exclusive loss of speaking, reading or writing, nor could they be divided into "motor" or "sensory" abnormalities. As the result of serial tests, he had attempted to show that no descriptive term can be found which covered all the functions disturbed in one or other forms of aphasia and kindred disorders of speech. For amongst these functions were many which were not usually associated with speech. He had

therefore adopted the name "Symbolic Thinking and Expression," because it represented that group of psychical activities which was most severely affected by unilateral lesions of the brain. Analysis of the clinical varieties of aphasia seemed to show that the various functions included under this term could be dissociated in different ways under the influence of organic injury. They did not correspond to different stages of dissolution or recovery: nor did they reveal directly the elements out of which language was built up. On the contrary they showed the components into which a highly complex set of psychical processes could be separated by destruction of certain portions of the brain. Something was lost which made perfect speech impossible, and the whole act assumed an abnormal form. Part of the manifestations were due to the negative aspect of the disturbance, part to the disorderly activity of function which remained.

(3) Before attempting to determine what portion of the brain when injured was associated with the different clinical affections of speech, it was necessary to discover by serial observations the nature of the psychical disturbance in each particular case. In the past attempts had been made to localize the anatomical site of the lesions responsible for "motor" or "sensory aphasia, "alexia" or "agraphia." But these *a priori* terms did not correspond to any specific functional categories discoverable in cases of these disorders of language due to a unilateral lesion of the brain. The fact that specific clinical forms of aphasia occurred showed that some lesions affected one aspect of the language mechanism more than another. He was prepared to enter on this question later, but was anxious that the present debate should be concerned with the nature of the loss of function produced by a unilateral lesion of the brain and with the clinical forms it might assume.

Dr. JAMES COLLIER: In opening this discussion upon the subject of "Aphasia," I must in the first place thank Dr. Head for the opportunity which he has afforded us and for his kindness in asking me to address you first. There has been for many years a growing revolution against the former conceptions of the nature of the speech function and the explanation of its defects, which has led to much bitter controversy, and has culminated in Dr. Head's bold endeavour to place this subject upon a sounder and truer foundation. It is very important in my opinion that we should have this opportunity of discussing this subject with him, and in this discussion there is no obligation that we should be restricted to the subject matter of his recent publications.

The necessity for this revolution was great. The literature is enormous and bewildering, and the types of aphasia multitudinous. Take for example Lichtheim's seven types based upon a conception of centres of localized function connected by isolated paths which only exist in theory, and with which types our clinical cases never truly correspond. Dr. Head would make a clean sweep of former conceptions and types of aphasia and introduce a newer conception of the functions of the cortex of the brain in speech, a more refined method of observation and a new classification. For the greater part I am in agreement with him. In some details I differ from him, while in others I am not in the position to express an opinion.

We have to bear in mind in dealing with the function of speech and its defects that we are considering the most highly evolved and complicated function of the brain, closely bound up with intellect itself, the mode of working of which is probably beyond any flights of human conception. And any analysis which we can make into this function must be of necessity crude and liable to misunderstanding. Conceptions of the coarse localization of function in the physiological substratum must, it seems to me, be erroneous, and any attempts to formulate the working of this intricate mechanism in diagram are fundamentally absurd.

I shall direct your discussion therefore to:—

(1) Your views upon the matter of localization.

(2) Your opinions as to the utility of retaining any of the time-honoured terminology.

(3) Would you retain any of the types of aphasia as true clinical types?

(4) Your views upon Dr. Head's method of examination and upon his classification.

The former conceptions of aphasia were based upon the most able clinical and pathological investigation of great men. They have served to teach us, and, so far as the localization of gross cerebral disease is concerned, they have led us to almost as great an accuracy in the regional diagnosis of local disease as we are ever likely to reach.

It is interesting to consider for the moment what were the grounds upon which the conceptions of isolated centres concerned with the registration of visual word images, auditory word images, and executive speech function were built up. It was from the study of comparatively large anatomical lesions, always subcortical as well as cortical, and generally more subcortical than cortical, and when such lesions were situated in such a position upon the confines of the cortical speech region as to sever more or less completely the usual paths of inlet to the speech region, either upon the visual side or upon the auditory side, or blocked the paths of exit upon the executive side, the most obvious of the speech defects resulting were failure of acceptance of speech on the visual side, word-blindness, or on the auditory side, word-deafness or failure of exteriorization of speech—motor aphasia and agraphia.

It followed that these limited regions—the angular gyrus, the mid-temporal region, and the third and second frontal convolutions—were labelled as visual word-centre, auditory word centre, and motor speech centre. I submit therefore for your discussion that when the conditions which have been termed "word-blindness," "word-deafness," "motor-aphasia," and "agraphia" occur, any one of them, as the most obvious result of a lesion of the brain, that they are the result of isolation of the speech region of the cortex upon the incoming sides and upon the outgoing sides respectively, and are not due to the destruction of local memory centres. It follows from the nature of the lesions that when these incoming and outgoing paths are damaged, there is often considerable damage to the cortex itself with the appearance of speech defects of a different order. Dr. Head would rid us of these terms, "word-blindness," "word-deafness," and "motor aphasia." Yet they are the terms which have been long in use, and while freely admitting that they may be fundamentally incorrect, physiologically inadequate and usually untrue clinically, yet these terms do roughly describe the most obvious features of many speech defects. They have proved useful for teaching purposes and will prove hard to replace.

In the Hughlings Jackson lecture Dr. Head doubts whether there are any such conditions as "pure alexia," "pure aphasia," and "pure agraphia," and in this attitude I am unable to follow him. For whatever conception we may have of the localization of function in the cortical speech region, we are bound to admit the existence of afferent paths to the speech region upon the auditory side and upon the visual side, and efferent paths therefrom by which speech is usually exteriorized, both spoken and written, and in these paths there is surely strict anatomical and physiological separation. In so far as these paths exist as definite anatomical tracts for a sufficient distance to allow of the isolated involvement of any one of them by a lesion, we cannot deny the possibility of such a lesion, nor that the resulting speech defect is more or less narrowly confined to one aspect of speech.

I hold that the clinical condition described by Dejerine as "pure alexia" or "pure word-blindness" and attributed to a lesion at the posterior limit of the speech area, which severs the connection of that area from the visual area, is one of the most certain and definite of clinical conditions. I have seen it many times and have seen the theoretical lesion verified on more than one occasion.

Similarly I see no reason why the conditions of pure agraphia and pure aphasia also described by Dejerine should not be true clinical conditions. Their acceptance does not affect our conceptions of the cortical functions of speech. There must be both physiological and anatomical separation on the way out of the paths by which spoken and written speech is exteriorized, and the possibility of isolated damage to such paths must be admitted to be possible. I have seen one case which during the early moments of his seizure was certainly a case of "pure agraphia." This was a man who was lying in the National Hospital with a fracture-dislocation of the back, who in the course of writing a letter to his wife was seized with a disability to write, to which he immediately had my attention called, and for the space of more than an hour, during which time his condition was investigated by myself and by Dr. Rennie, now of Sydney, he presented a defect which exactly corresponded to "pure agraphia." After which his lesion extended widely, and proved rapidly fatal. We found in this case a subcortical lesion beneath the lower frontal convolutions associated with an extensive thrombosis of the Sylvian artery, and I think it is not too much to claim that the commencement of the subcortical lesion was in such a position as to block the outgoing path for written speech, and did actually produce a condition of "pure agraphia." I agree that pure "word-deafness" does not exist as a clinical entity, and I would explain this fact in that it is unlikely for any lesion to sever the auditory path entering the speech region of the cortex without interfering grossly with that cortex both anatomically and physiologically. Yet there are cases which all of us must have seen in which lack of acceptance of spoken speech stands out in undue contrast with the acceptance of visual speech, and I would attribute this isolation of function not to any separate location of function in the speech cortex, but to blocking of the auditory afferents.

I presume that the chief paths entering the cortical speech region do not end abruptly, nor do the outgoing paths begin abruptly, but rather gather in their course, and that therefore the element of blocking must be an important factor in the production of the speech defects under consideration.

There is another consideration in connection with the afferent paths which I deem of great importance, especially in connection with those speech defects which Dr. Head terms "syntactical." When we speak aloud we have an immediate recognition of what we say by means of the auditory path, which allows of analysis of the correctness and fitness of what we have said and of its correction if faulty. This backlash, if I may be allowed the expression, is of great importance during the learning of speech in any form, and it surely must be a great factor when we speak, in keeping the physiological activity of speech and thought at a high level.

There is also another backlash in the way of the sensations accompanying the movements of the executive mechanism, but this plays but a very minor part in the speech of normal persons, though since in the deaf-mute who has learned to speak by lip reading it is his only guidance both in the learning of his speech and in its execution, this factor must not be forgotten.

There is of course a similar constant visual appreciation and correction in writing.

Now, when on account of the lack of acceptance of spoken speech this immediate consciousness of the correct execution of speech fails, surely this must throw the mechanism of thought and speech into dire confusion. I submit that this is the essential cause of those defects of speech which Dr. Head groups as "syntactical," and certainly of such conditions as voluble jargon-aphasia. I shall refer to this subject again later on.

Dr. Head in his Linacre Lecture seems to favour some vague separation of function, some very general localization of functions in the cortical speech area which he does further comment upon. If by this he means a gradual passing over from behind forwards from receptive functions to executive functions, I am inclined to agree with him. I take it that he would expect "verbal defects" from lesions farther forward than those productive of "nominal" and "semantic" defects. If on the other hand he means any separation of localization for visual and auditory speech functions, then I disagree.

It seems to me that the function of visual speech, acquired at a much later age than is auditory speech, tacked on as it were to an already perfect and deeply impressed function of auditory speech, learnt by means of an already well versed auditory speech function, must occupy the same physiological substratum as does the auditory speech function, and is separately localized only in so far as its chief afferent and executive paths are concerned.

I submit for your discussion, that, as the result of lesions of the cortical speech region in any of its parts, there is likely to be depression of its physiological activity as a whole, and that the expression of this depression may be a major impairment or loss of its less deeply impressed functions. For the speech functions are not equally impressed nor equally stable. The auditory speech functions both upon the receptive and upon the expressive side are in most people much more deeply impressed and more stable than are the corresponding visual speech functions. And I submit that this accounts for the impairment of loss of visual speech recognition which almost always occurs in lesions of the anterior regions of the speech cortex, and that it accounts also for the major loss of the visual speech functions which is the rule in lesions situated farther back.

I have in two cases observed the practically complete recovery from aphasia in polyglots who were afflicted with syphilitic vascular disease of the speech region, and in each case recovery was much more rapid in the more deeply impressed native tongue, and less rapid and less perfect in the language more recently acquired, and less long used. And when speech was practically recovered so far as the native tongue was concerned they still presented gross defects in the foreign tongue. It would be fair to say that at a certain stage of recovery they were aphasic in one language and not in another, and this must surely be explained by the dropping out of the less impressed function when the activity of the speech functions is lowered, as the result of disease.

Dr. Head calls particular attention to the highly important and certainly correct principle that the alterations in speech function which result from lesions of the brain are not in terms of the primary elements out of which speech has been evolved, nor do they reveal the elementary basis of the acts of speaking, reading and writing, but that they are in terms of the complete and highly developed function, which fails progressively from its highest to its lowest with increasing degrees of damage.

Though in infancy we learn our speech in terms of words and short phrases, and our writing in terms of letters and words, yet when speech is highly developed we speak and receive speech in a running pattern of sounds of which the individual elements are disregarded. We do not in reality speak, hear, read or write in words, but in a running pattern comparable and often inseparable from the running patterns of thought. It is the pattern which is exteriorized and accepted as a whole. The individual words of which this pattern is made up singly convey little or nothing of meaning unless we put them into another pattern by parsing them. The running patterns of speech are of great importance in the consideration of speech defects. In their course they reinforce and raise the level of activity of speech and thought, and they have an important momentum. The common example of the aphasic who is unable to use a particular word on command, and yet produces it without effort in the run of the pattern by which he is expressing his inability, or trying to talk round the subject, well illustrates this point. In setting a speech pattern going he gets a run at his difficulty just as in taking a running jump one gathers the necessary momentum to overcome the difficulty. Deformity and distortion of these patterns are common phenomena among speech defects, and the difficulty to set them in orderly running is fundamental.

Sometimes one is surprised in examining a case in which speech acceptance is seriously impaired, to find that the patient will gather considerable meaning from a phrase when he does not do so from simpler forms of approach. He accepts something from the pattern when simpler forms of communication do not appeal to him.

Dr. Head has laid before us in the Linacre Lecture a scheme for the examination of patients suffering from defects of speech which seems to me in every way admirable and worthy of universal adoption. It provides for the examination of the speech functions in the varying states of their incapacity from the lowest to the highest. The serial tests are calculated to rouse into activity depressed functions which otherwise give no sign of their existence, as for example the patient who though unacceptive to visual speech in any ordinary way matched a colour with its printed name, thereby showing that some dim function of visual speech was rousable by the combined presentation of the colour and its name. Of especial interest as arising from the serial tests is the proposition that inasmuch as an act requires verbalization for the clear recognition of its nature, the act tends to fail in execution if verbalization is defective. This proposition emphasizes the importance of verbalization in the concept. It is so far as I am aware a new proposition—at any rate it is new to me and seems to me to be of far-reaching importance in the interpretation of speech defects.

The serial tests are excellent, the difficulty comes with the interpretation of the results. The classification of speech defects into "verbal" defects, "nominal" defects, "syntactical" defects and "semantic" defects requires careful consideration. Dr. Head's position is clearly defined. He starts with the conception of a region of the cerebral cortex concerned with speech functions which habitually acts as a whole and not in terms of isolated localized functions. This position seems to me correct and unassailable. He makes an analysis from the clinical side only and commits himself to no opinions of anatomy and localization, but deals entirely with objective physiological defects in his attempt to elucidate the manner in which the speech function breaks up as the result of lesions of the brain. Whatever views we may hold individually upon the nature of the speech functions, such an analysis is not controversial. It can offend no one. It certainly is very valuable. It rests upon a sound scientific basis. In making this analysis he used specially picked cases. Selected firstly because of slight and partial lesions which leave a great part of the anatomical substratum of speech in a relatively physiological condition, and therefore are much more suited for analysis in that they yield much more information. Selected secondly because from the nature of the lesion the cortex of the brain may be deemed to have been particularly affected, though this selection may give rise to doubt as to whether the lesion in any given case was mainly cortical; it is open perhaps to less objection in this respect than are the vascular and other gross lesions in civilian cases upon which great theories have been built up. And lastly selected because they were suitable for the analysis—all cases are excluded in which images are lost. Such an arbitrary selection must be allowed if we are to get a clear issue and avoid confusion. The results of the analysis are recorded in minute detail. There is no attempt at explanation, and no theory is raised upon them. Localization is not considered as this is an anatomical matter. The results are in the present state of our knowledge useful in enumeration only. The classification is a list of symptoms which from their nature tend to fall into four groups, and the patients tend to fall into four corresponding groups.

Dr. Head submits that these groups are the expression of the manner in which the speech function breaks up as the result of the slighter degrees of damage to the cortical substratum subserving that function. Dr. Head's position does not admit of much contention or controversy. It does not in any way subvert or undermine the correctness of the rough methods by which all of us are accustomed to localize gross cerebral disease with accuracy when the speech region of the cortex is involved. It strikes fundamentally at former theories both of the nature of the speech function, and at the localization of separated functions—not many of which I submit have been truly applicable to clinical conditions nor theoretically satisfactory.

Comments upon his division of symptoms and his classification of cases into the four groups cannot be adequately made until we have applied the serial tests to our suitable cases and made our own deductions therefrom in the light of this classification. Whether this grouping does really represent the manner in which the cortical speech function may disintegrate is to me uncertain, for by no method of introspective analysis, no consideration of the development of speech functions, and by no anatomical or physiological considerations can I see why it should do so.

I shall conclude these remarks in referring to two points in connection with his classification which I deem of considerable importance. In connection with verbal aphasia it is quite refreshing to see it laid down with emphasis that deformity of words in the way of mispronunciation, omission and misplacement of syllables—abnormal word formation, in fact—is characteristic of this condition, and further that the same deformity that occurs in the spoken words is met with also in the written words, proving the inseparability of these two functions. This is a clinical fact which has been neglected or passed over by so many writers upon aphasia. Sometimes this deformity is so great as to make the speech almost irrecognizable and in a way a jargon-aphasia. I have been in the habit in the past of referring to this condition as "anterior" jargon-aphasia from the forward situation of the lesion which is usually responsible for its production. It is of course instantly distinguishable from true jargon-aphasia. I am not sure that the essential rhythm of word and of phrase in this condition is always intact as Dr. Head thinks. I believe that considerable defect of rhythm may occur and that the phrase may be deformed as a whole just as the word is deformed.

In syntactical aphasia as a result of damage to the speech region of the cortex, I find myself for the first time in the course of these remarks quite opposed to Dr. Head; it is his meaning that such a condition can arise from a lesion of the cortex. True jargon-aphasia is a thing quite of itself. It is the only condition among speech defects in which overaction is manifest. The volubility which is characteristic of this condition is extraordinary and seems to increase the more severe the jargoning of the speech becomes. Even from the first there may not be that general depression and difficulty of arousing the speech functions that is seen in connection with other forms of speech defect. The intelligence and quickness of apprehension of all uncomplicated cases of jargon-aphasia even in its severest degrees stand out. The condition seems to me that of a function severed from control and without guidance. I must admit that I have not had the opportunity of examining cases with such mild degrees of this condition as has Dr. Head. I have never in any case of this nature seen so great a degree of speech acceptance upon the auditory side as some of his cases had. In all my cases there has been severe blocking upon the auditory side.

I may here refer to two cases almost identical in nature which were under my observation and which were of so striking a nature as perhaps to have biased me unduly in my conception as to the nature of jargon-aphasia. These were both men who at their work were seized with a slowly oncoming jargon-aphasia which in the course of a few hours rendered speech incomprehensible. There was no ictus with the onset, no affection of intelligence, and these men did not even stop their work. They were brought to me within a few days—the one by the police for making a disturbance in the street, the other by his wife, as being mad. They were both uncomplicated cases of severe jargon-aphasia. The one was absolutely non-acceptive of speech either upon the auditory or upon the visual side, and the other was non-acceptive upon the auditory side, but appreciated visual speech to a considerable extent. One of these patients presented optic neuritis, and the other soon developed it. Both of the cases showed on pathological examination a well-defined tumour undercutting the posterior and mesial aspects of the temporal lobe, comparatively far from the cortex which was nowhere involved. The tumours were in such a position as might reasonably be expected to sever the auditory afferent path to the speech region in both cases, and in the first case the tumour curved round towards the cortex in the angular region, and is likely to have severed the visual afferents.

The striking points about these two cases were :—

The onset of jargon-aphasia as the initial symptom.

Its rapid development to an extreme degree.

The lack of acceptance of auditory speech.

The peculiar subcortical position and discrete nature of the lesions which nowhere involved the cortex.

It was difficult to regard these cases in any other light than that of isolation phenomena in which the auditory balance and rhythm which is so essential a part in the production of speech was lost owing to the interruption of afferent paths to the speech region of the cortex, by which speech exteriorization is habitually guided. I submit that it is the lack of the immediate impressions of correct execution which we receive when we speak, both with our ears and from the muscular movements of speech, which is responsible for the condition of jargon-aphasia. It is the expression of a function which has lost control and which has "gone mad" from isolation.

Surely these cases of jargon-aphasia are primarily unaware of their defect of utterance by reason of lack of auditory acceptance, some of them partially perhaps, but the majority of them deeply. They do not show the embarrassment and loss of confidence that such defects, if recognized, would certainly cause, though they may show some embarrassment and emotion from lack of appreciation of what they are trying to say in their audience. It is true that in one of Dr. Head's cases there was a greater degree of auditory acceptance than I have ever seen in a case of jargon-aphasia, but I gather that all of them were more or less defective in this way, but to what degree he does not make very clear.

Visual speech and writing are almost always affected to a less degree than auditory speech in cases of jargon-aphasia, and this I attribute to preservation of the visual afferents. One of Dr. Head's cases seems to show that correct writing can occur without verbalization—I refer to the case who on being shown a matchbox uttered a three-syllable jargon and promptly wrote the word matches correctly. This is a very interesting and unusual fact, for writing is usually strictly dependent upon verbalization, and when verbalization is imperfect the writing shows exactly the same defects as does speech. I agree with Dr. Head that verbalization is always very imperfect, and in the more severe degrees of the condition writing is almost as much affected. Now, this relative escape of the visual word function is surely the reverse of what occurs in cortical lesions, in which, I believe, the visual word function always shows a major defect compared with that of the auditory word function, and I use this as an argument against the cortical origin of jargon-aphasia.

Further, jargon-aphasia is a comparatively rare condition. It is hardly ever met with in the common lesions of the temporal region from vascular disease of the Sylvian artery where the temporal cortex is likely to be involved. I think also that when jargon-aphasia is due to a progressive lesion, an extension of the lesion to the temporal cortex causes the jargon-aphasia to lessen in volubility and finally to disappear.

I submit that an intact cortex is necessary for the appearance of this form of speech defect, and that it is a pure type of aphasia, and like the other pure types of aphasia is referable to an isolation from auditory afferents and to a subcortical lesion.

SIR JAMES PURVES STEWART: We are so accustomed to receive original and stimulating thoughts from Dr. Head that whenever he brings us something fresh many of us tend to be prejudiced in advance in his favour. This, of course, being a mere feeling-tone, partly thalamic, and not a process of cortical reasoning, is just as wrong as it would be if we were to start prejudiced against him.

At the outset let me say that Dr. Head's conception of aphasia is full of instruction to us all. No one will venture to cast any doubts on Dr. Head's clinical observations. Every one of us must be grateful for the new scheme of investigation which he has presented to us and for the various interesting clinical facts which this scheme has brought out. We have studied with interest his proposed new classification of aphasia into four varieties, viz.:—

(1) Verbal aphasia. Defective word-formation, both in internal and external speech.

(2) Nominal aphasia. Defective use of nouns, both spoken and written.

(3) Syntactical aphasia, with want of coherence and a tendency to talk jargon.

(4) Semantic aphasia. Deficient comprehension of the full significance of words and phrases. Although the patient can speak and write, he cannot formulate a general conception but only the details of which it is composed.

This new classification is of the highest psychological value. But there seems to me no adequate reason why we should necessarily throw overboard all our previous clinical experiences, or our division of aphasia into two great classes, viz.:—

(1) *Psycho-motor aphasia*, aphasia of expression, or verbal apraxia.

(2) *Psycho-sensory aphasia*, aphasia of comprehension, or verbal agnosia.

More especially as these two great clinical divisions correspond to well-established anatomical facts.

One thing we note in Dr. Head's recent paper is that he abstains, so far, from giving us particulars of the anatomical site of the brain-lesion in a single one of his cases.

May I be allowed, as an ordinary clinician, to relate a few clinical examples of aphasia. All except one have been selected, like Dr. Head's cases, from young men suffering from war-lesions of the brain.

Case 1.—W. M., aged 55. Lower frontal lesion.

This patient was admitted to the Westminster Hospital with signs of acute cardiac failure due to old aortic disease. He had no paralysis of the face or limbs and was able to speak fluently. His pulse was almost imperceptible and the heart sounds were almost inaudible, forty-eight per minute.

Twenty minutes after his admission to the ward, he suddenly became speechless and his face became asymmetrical. His intelligence was unimpaired. He was able to understand and execute all sorts of verbal commands, to put out his tongue, to place his hand on any part of his face or head, to move the right or left thumb by bending it half-way or straightening it out, as requested. But he was unable to utter any word whatever. The right face was weaker than the left. There was no weakness of any limb, but the right plantar reflex was extensor in type and the right abdominal reflex was absent.

Two days after admission he died, with symptoms of increasing cardiac weakness, cyanosis and rhythmic breathing of stertorous type.

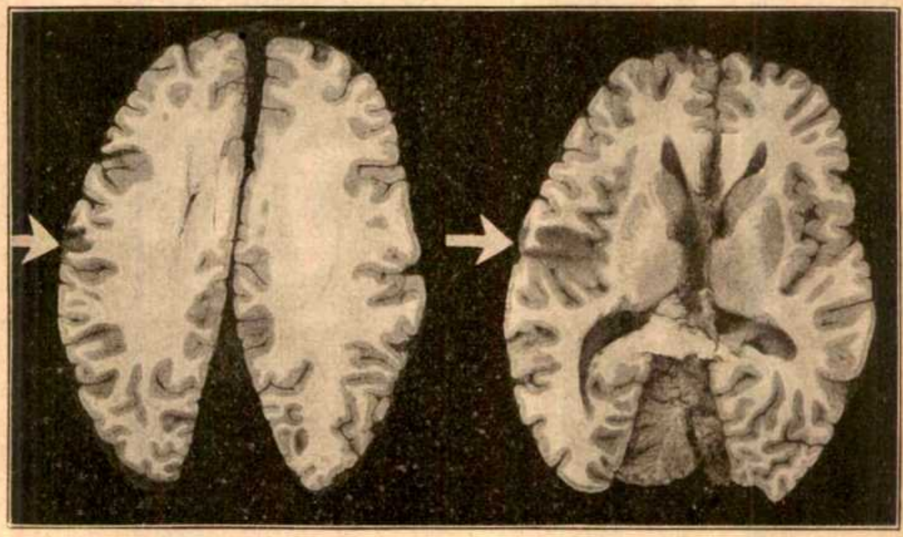


FIG. 1.—Left lower pre-central lesion.

At the autopsy, there was an old syphilitic aortitis, and just above the aortic valves was a large tag of thrombus about an inch long and one-eighth inch thick.

The brain, after hardening, showed a small area in the lower third of the left precentral gyrus, in which there was a dark marbled appearance, due to embolic blocking of a small cortical arteriole (see fig. 1). This lesion extended deeply into the operculum, but did not affect the insula, nor the basal ganglia, i.e., Marie's "lenticular zone" was unaffected. There was no other lesion elsewhere in the brain.

This is an uncomplicated case of psycho-motor aphasia. Probably Dr. Head would classify it as a verbal aphasia.

Case 2.—W. T., lieutenant, aged 24. Mid-Rolandic lesion, pre- and post-central.

The patient was wounded by a ricochet bullet in the left parietal region. He was knocked down, but not unconscious. His speech at once became jumbled. After a quarter of an hour he noticed paralysis of the right arm, and after about four hours the right leg also became paralysed.

A week later he was trephined, and two pieces of bullet were removed from the surface of the dura. The dura was not opened.

The patient states that after his injury he could not get out his words at all, nor could he understand things properly.

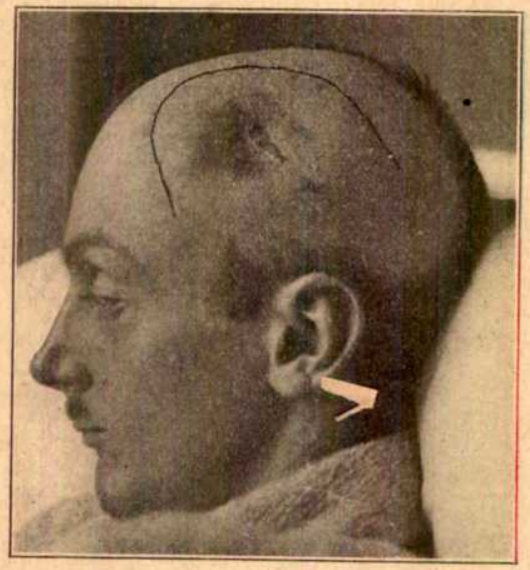


FIG. 2.—Left pre- and post-central wound.

On examination, five weeks after the original injury, he was highly intelligent and had no evidence of word-deafness or word-blindness. He understood and executed accurately verbal requests and also written requests, e.g., "Close your eyes and then put out your tongue."

He was able to write spontaneously and to dictation short sentences with his left hand, copying accurately, but occasionally making mistakes in spelling to dictation, e.g., "hopsital" for "hospital."

He recognized all objects shown to him and named them correctly, but tended to miss out the initial consonant, e.g., "'overeign, 'illing, 'atch-box." But he repeated words and phrases correctly to dictation. He added up columns of figures fairly accurately and read the newspapers intelligently.

The right face was slightly weaker than the left. There was severe paralysis and spasticity of the right upper limb and slight weakness of the right foot. There was astereognosis of the right hand and foot. With the right hand he recognized an object like a coin as hard and round, but could not name it. The deep reflexes were increased in the right upper and lower limbs, with right-sided ankle-clonus. The right plantar reflex was extensor in type and the right abdominal reflex absent.

A month later he had improved a great deal. He now only hesitated very slightly, chiefly with initial sounds. Sometimes he misplaced syllables, but chiefly when excited. He felt himself mentally brighter, but thought he was a little slower than before his injury, e.g., when counting money.

He understood everything said to him, and executed accurately written commands. He copied accurately from printed to written letters and wrote to dictation (left hand) without making spelling mistakes. He added up figures, both simple figures and £ s. d., e.g.,

£2 17 9
8 13 4
£11 11 1

He repeated words dictated to him, but sometimes mispronounced syllables, e.g., said "February twenty-fith." He named objects shown to him accurately, also read aloud accurately from a book.

The weakness and astereognosis of the right upper limb were still severe, but he was now able to walk, dragging the right leg.

We clinicians would call this an example of psycho-motor aphasia. Dr. Head would probably classify it as a verbal aphasia.

Case 3.—D. M., aged 31. Inferior frontal lesion.

This patient was seen at Salonika. Eight days before I saw him he was admitted to hospital unable to speak, but able to understand what was said to him and able to write with his right hand, jumbling his words in writing.

He had a depressed fracture of the skull in the left upper temporal region (see fig. 3) over the region of the lower end of the Sylvian fissure, apparently from a glancing shrapnel wound. There was a large lacerated scalp wound, and the fracture was found to have fissures radiating backwards for a short distance. The depressed piece of bone was removed by trephining. The dura was intact and pulsated normally. It was not incised. This operation was performed four days after his original wound.

A week after the operation his temperature was normal; there was weakness of the right face with deviation of the tongue to the right side. There was no weakness or anæsthesia of the upper or lower limbs. The knee-jerks and ankle-jerks were brisk and equal; the plantar reflexes were both flexor in type and the abdominal reflexes were normal on both sides.

He had no word-deafness nor word-blindness. He understood and executed verbal requests smartly, also a written order to put out his tongue.

He spoke with some difficulty. When asked his name he said: "Duncan M-M-M-M-enzies." He could not name a postcard, a pin, or a safety-pin. He called a fork a knife, but at once recognized his mistake, and when shown different objects, including a knife, fork, and spoon, and asked: "Is that a knife?" always said "Yes" when the correct object was indicated, and "No" when it was not so. He wrote a postcard to his parents, writing their address correctly, but repeating several words. His message ran: "I am getting only, only, yours, Duncan."

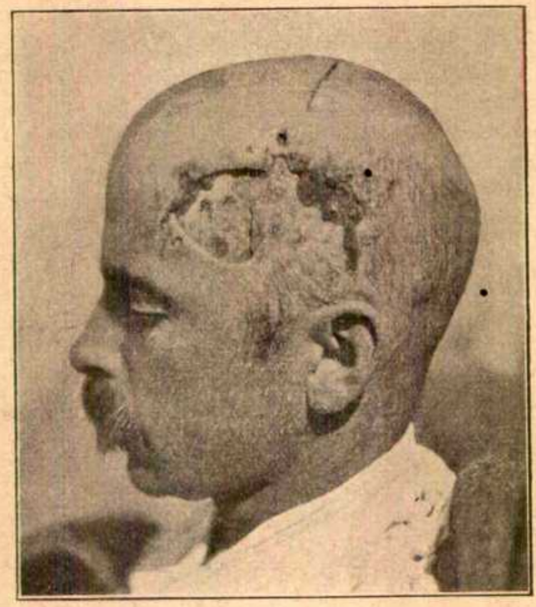


FIG. 3.—Left inferior frontal wound.

A week later he spoke better, but slower than normally, occasionally confusing his syllables. He called a coin a "Greek fenny," and a safety-pin a "sapety-pin."

A week later he no longer jumbled his syllables, and, save for a certain amount of slowness, his speech was normal and fluent.

This again is a fairly straightforward case of psycho-motor aphasia. Dr. Head might call it verbal aphasia or nominal aphasia, or both.

Case 4.—W. Y., aged 19. Temporal lesion.

He came into hospital without any available history, but he had probably been wounded four or five days previously at Gallipoli and brought straight to Malta, where I happened to see him.

There was a superficial entry-wound three inches vertically above the tip of the left pinna and an exit-wound half an inch above the posterior end of the left zygoma (fig. 4). The cranium was not fractured, and X-rays showed no abnormality.

He had no weakness or anæsthesia of the face, trunk or limbs. The reflexes, deep and superficial, were normal and equal on the two sides. There was no limitation of the visual fields.

He understood and executed simple verbal requests, but very easily became confused, e.g., when told to place his right hand on his left ear. He read aloud slowly and accurately, but did not understand what he read, nor did he make any attempt to execute written requests which he read aloud. When shown the statement $\begin{array}{c}2\\ \underline{2}\\ 5\end{array}$, however, he said, "That's wrong."

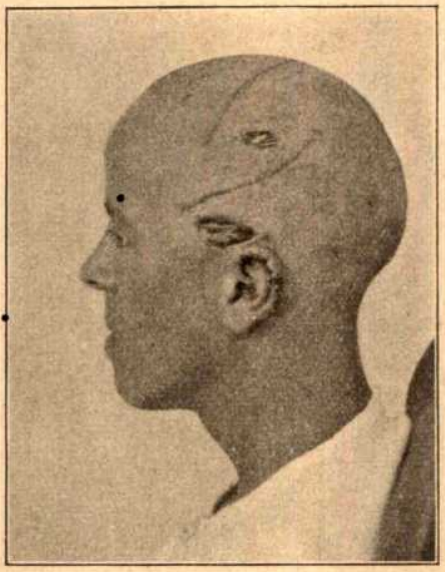


FIG. 4.—Left temporal wound.

He spoke slowly and with difficulty, but could name objects shown to him. When speaking he occasionally jumbled up syllables.

This is a case of psycho-sensory aphasia, with word-deafness, paraphasia and word-blindness. Dr. Head would probably call it a combination of nominal and semantic aphasia.

Case 5.—C. E., captain, aged 22. Temporo-occipital lesion.

Patient had a penetrating wound by a shrapnel bullet. The entry was above and behind left ear, over the posterior end of the first temporal gyrus (see fig. 5). There was no exit-wound and an X-ray showed the bullet lodged ½ in. to the left of the middle line, just above the occipital protuberance, i.e., near the tip of the occipital lobe.

Four days after his injury he developed meningitic symptoms with general epileptiform fits. The wound was laid open and several pieces of in-driven bone were removed from the brain. The fits subsided a fortnight after his injury.

When examined three weeks after the injury, he had a voracious appetite, possibly attributable to his temporal lesion. The wound was soundly healed.

Five weeks after his injury he had no weakness of face or limbs, no anæsthesia, normal deep and superficial reflexes, but a right upper quadrantic hemianopia in both visual fields, due to lesion of the lower lip of the left calcarine cortex.

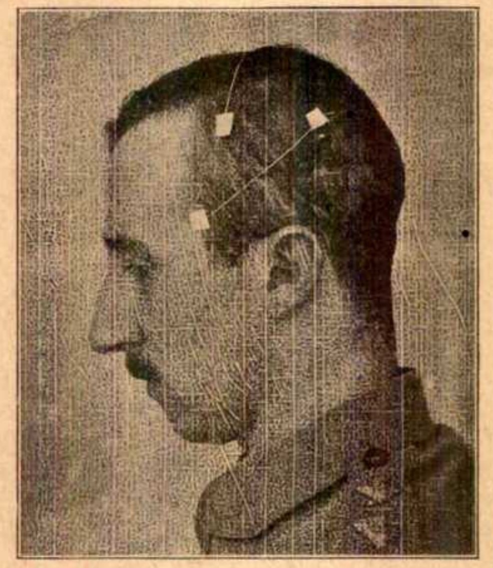


FIG. 5.—Left temporal wound, with bullet in left occipital lobe.

He understood and executed simple verbal requests, but had difficulty in executing complex requests, e.g., to place the right middle finger on the left ear. He executed written requests, wrote fluently, but occasionally made spelling mistakes, e.g., mis-spelt "Malta" as "Matla." He copied accurately from printed to written letters.

In speaking he had slight hesitation in naming objects but ultimately succeeded. He stumbled over syllables in words like "Gallipoli peninsula." He spoke fluently as a rule.

Here again the aphasia was of psycho-sensory type, with slight word-deafness and paraphasia, or mal-construction of words. Dr. Head would probably regard it as a case of generalized loss, not belonging to any one category.

Case 6.—J. C., aged 25. Temporo-parietal lesion.

Patient was admitted to hospital without any history, but with a recent operation-wound, apparently about a week old, on the left side of the head, including a trephine gap 2½ in. above and 1 in. behind the left external auditory meatus (see fig. 6). There were also an entry and an exit-wound, one at the left parietal eminence, the other just below the centre of the left zygoma. Which was which, we could not decide.

He had no weakness of the upper or lower limbs; there was doubtful paresis of the right side of the face. He had no hemianopia on rough test and no cutaneous anæsthesia or analgesia. The knee-jerks and ankle-jerks were normal. The right plantar reflex was extensor; the abdominal reflexes were brisk and equal.

He had considerable difficulty in understanding and carrying out even simple verbal requests. When shown written commands he spelt out the letters correctly but made no attempt to carry out the instructions. He wrote with a pencil, writing his name spontaneously and copying from printed to written letters. When shown the statement $\begin{array}{c}2\\2\\\hline 5\end{array}$ he did not recognize anything wrong.

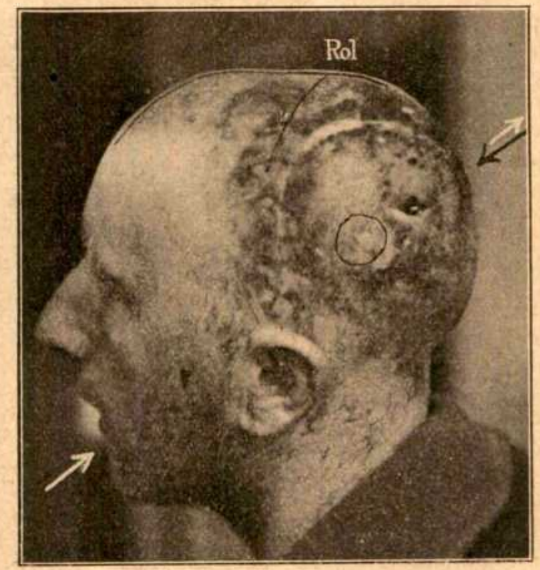


FIG. 6.—Left zygomatico-parietal wound.

He could not repeat words dictated to him nor could he write to dictation even simple words like "hospital."

When shown scissors, he could not name them, but said, "I know what they are for," and made scissor-movements with his fingers. With a pencil, he said, "I know what it is," but could not name it.

A week later the word-deafness was less marked. He could not write to dictation but made mistakes in spelling, e.g., "arterly" for "artillery." He could repeat words dictated to him but could not name objects, such as scissors or a pencil, saying, "I don't know the name of it, I forget."

This case would probably be regarded by Dr. Head as being of the "nominal" variety. But on clinical and anatomical grounds it is a straightforward case of psycho-sensory aphasia.

I might have quoted many other cases of more profound aphasia, due to widespread lesions, but the cases which I have related have been selected because of the relative simplicity of the brain-lesion in each case.

We find then, in cases of psycho-motor aphasia, or aphasia of expression, a lesion in the anterior or psycho-motor area of the brain, whereas in psycho-sensory aphasia, or aphasia of comprehension, the lesion is situated in the psycho-sensory speech areas, whether psycho-auditory or psycho-visual. In both classes of case there is always a certain amount of intellectual disturbance, the equilibrium of the speech areas being rendered less secure, just as a table becomes unsteady, whether one of its anterior or its posterior legs happens to be broken.

We are familiar with Pierre Marie's view that all aphasia is merely an intellectual deficiency, due to disintegration of some part of Wernicke's zone. He claims that the essential fact of aphasia, of whatever variety, is insufficient comprehension of speech. We also know that Marie discards a special motor centre for speech and denies to Broca's convolution any special rôle in the function of speech, except in so far as it contains motor centres for the lips, tongue and larynx. He explains motor aphasia as mere intellectual deficiency *plus* anarthria, and refers this anarthria to a lesion of the lenticular zone (comprising the lenticular nucleus and its surrounding white matter).

But motor aphasia is not a mere paralysis of the lips or tongue; it is a true apraxia, due to amnesia of the movements of articulation. Moreover, if articulative difficulty were the cause of the speech loss, it ought to render the articulation of all words difficult. Now we know that most aphasics, however profoundly speechless, are not wordless. They usually have a "recurrent utterance" of some sort, and this recurrent utterance is articulated perfectly clearly.

It would, therefore, seem that the psycho-visual, psycho-auditory and psycho-motor speech centres do exist, and that lesions of these centres, disrupting the cerebral mechanism of speech, are the cause of our well-defined clinical types of aphasia.

Dr. Head by a series of ingenious and elaborate tests has arrived at a new, useful and philosophical basis of classifying aphasias. But this does not necessarily mean that other classifications may not also be correct. Just as one observer may classify a crowd of people into men, women and children; whereas another may classify them into Englishmen, Scotsmen, Irishmen, Welshmen and foreigners; or another again into soldiers and civilians. It all depends on the angle from which the facts are viewed. Dr. Head has opened a new window into the chamber of aphasia. But this does not compel us to close up all the other windows, especially the anatomical and pathological windows, through which so much light has been admitted in the past.

Dr. KINNIER WILSON: It is with a certain amount of hesitation that I venture to express my views in regard to Dr. Head's recent contributions to the difficult subject of aphasia. Perusal of his papers is not enough, perhaps, to justify criticism or even to arouse full appreciation; it is evident that the student of the question must himself utilize some or all of Dr. Head's ingenious tests for the aphasic in order to be in a better position to pass comment on his conclusions.

Nevertheless, certain general considerations seem worthy of a little attention.

It is clear, in the first place, that Dr. Head's contributions must be viewed solely from the clinical standpoint. The question of localization is not raised, with the exception that he speaks throughout of "unilateral lesions," presumably in the left hemisphere. As there is no record of any pathological investigations, one cannot even be sure that the lesions have been strictly unilateral. Incidentally, I cannot agree with him that the "G.S.W. head" type of case is better for the study of the subject than vascular cases, for he himself admits that gunshot wounds tend to cause greatest damage on the surface of the brain, in addition to which there is the factor of commotio in all such patients. The fact that small hæmorrhages may catch projection fibres where they converge or diverge, is, in my opinion, much less important for the subject of aphasia than that association—i.e., transcortical, fibre systems should be involved, and these are obviously more likely to be impaired in cases where the damage is mainly cortical. The ideal case is neither the tumour nor the gunshot wound case, but that with isolated vascular lesion, the exact position of which there is opportunity to establish post mortem. Such cases are few and far between, but that increases their value when they are obtained.

Ignoring, however, the pathological side of the question, and the whole matter of localization, one may attempt to form some judgment on the clinical side of Dr. Head's work.

(1) We must be in agreement with him when he shows with clarity that the various types of aphasia are produced by dissociation of a definite mental process, which he designates symbolic thinking and expression. No one can suppose that aphasia is due to loss of motor or sensory power; if a patient has his speech musculature paralysed he is anarthric, or if paresed, dysarthric, but these are phenomena of a lower (middle) level, and the same is true of ordinary cortical blindness or deafness; these are not elements in aphasia Dr. Head holds, further, that aphasia is not due to "diminution of general intellectual capacity," as Dejerine and others have also held in opposition to Pierre Marie, and some of my own cases may certainly be taken to bear this out. Not a few strictly limited cases of aphasic disturbance are on record to substantiate the truth of the statement. It appears to me, nevertheless, that some of Dr. Head's tests are tests of intellectual capacity, or at least of eupraxia, rather than of speech activity in the ordinary sense; in particular, his mirror test. Accepting Dr. Head's evidence from a large number of his cases of aphasia, that the patient has difficulties in imitating movements made by the examiner when the latter faces him, but can perform them easily when they are seen in a mirror, I am not able to follow the suggested explanation, that in the former group the imitative movements are "accompanied by more or less internal verbalization," whereas in the latter "no verbal symbol intervenes between the reception of the command in gesture and its execution." It seems to me largely a question of automatism. All of the patients are certain to have performed movements in front of a mirror every day of their life, so to speak, e.g., in shaving, hair-brushing, &c., and such types of movement become automatic; whereas, I do not suppose a single one of them had ever practised direct imitation movements. Their difficulty, therefore, I take to be one of practice and not of internal verbalization, and this is borne out by Dr. Head's statement that many improved very rapidly in this faculty. The test can, I think, be fairly set down as one of "glegness in the uptak" which varies enormously with the individual. Besides, not a little evidence is accumulating to suggest that automatic processes, automatisms, are located in the *right* as opposed to the left hemisphere. It may be remembered that Hughlings Jackson himself held that the right was the more "automatic" of the two; hence, one should not expect the same impairment of acquired automatisms in *left* hemisphere lesions, where one may presume the latter are located in Dr. Head's cases. The point, however, can only be alluded to in passing.

Again, the author maintains that aphasic defects are not due to destruction of images, showing by examples that many aphasics have not lost their images at all. I do not think the common view of aphasia is that in its different forms different types of image are necessarily *destroyed*, but that the patient is not able, owing to his organic disease, to awaken them when he wishes to. Thus a patient of my own after a stroke became absolutely mute. His state was one of complete motor aphasia; he had no words, not even "yes" or "no," and had apparently lost all his motor images, but on the night of the destruction of the Zeppelin at Potter's Bar he was a spectator of the wonderful sight and called out "Hallelujah," which word he has never since repeated in spite of innumerable efforts. The "engram" could not have been destroyed, and while it may be impossible as a rule to prove that images are not destroyed, for the practical purposes of speech they are "lost," and it seems to me to matter little, in a way, what has really become of them in such cases. Probably the question is largely one of the extent of the pathological lesion, and as in the majority of instances images can be shown to be retained, the lesions cannot be "knock-out" ones. After learning that in Dr. Head's strongly expressed view the affections of language from (presumably) unilateral lesions "are not based on a destruction of images," one is a little surprised to be told subsequently that he expressly excludes cases of aphasia where the patients "showed evidence of definite destruction of sensory images." It is admitted, then, that some cases are image-less. Of course, in the mind the concept of an object, say an orange, is so compact of differing linked images that it can be aroused in consciousness by stimuli from one sense avenue when others fail; Dr. Head has well shown in one of his routine tests how an aphasic can recognize an object by touch, say, when visual images fail him, and so on. Only in the most widespread cases of cortical impairment, therefore, would one expect destruction of images, and since the sensory components of the synthetized complete idea of an object are, I hold, located in widely separated cortical fields, it naturally follows that destruction of images on any wholesale scale must be a rarity. For example, too many cases have been classed as agraphia because the patient cannot write with his right hand as a result of a cerebral lesion, but too rarely is the test made with the left; for that matter, an agraphic patient, so-called, might conceivably still be able to write with the pen in his teeth, or held by his toes.

(2) This leads to a second consideration, which is, that Dr. Head holds that the familiar terms "aphasia" itself, "alexia," and "agraphia,"

should never be employed, because the disorders of language produced by a unilateral lesion of the brain are never "*exclusive* affections of speech, reading, or writing."

In another place he says—and I wish to draw special attention to the statement—the function which is disturbed by such lesions of the brain "cannot obviously be comprised under the headings of speaking, reading, and writing; for not only may the loss of power to carry out any one of them be partial, but the disturbance extends beyond their limits and affects other mental activities."

Now it may be granted at once that "pure" cases of these disorders are doubtless rare, that mixed varieties are the rule; and, for the sake of argument, one may say with Dr. Head that "inability to write is always associated with some other loss of function." It is, nevertheless, a clinical fact that one or other defect, in speaking, reading, or writing, may be *by far the most prominent* feature of a given case, so that while, for example, speaking and reading may be for practical purposes intact, writing may be all but impossible, as in a good case published in 1903 by Stanley Barnes, and similarly with alexia (combined with hemianopia) from lesions in the distribution of the posterior cerebral artery, as in a striking case of my own, and with pure motor aphasia as excellently shown by Dejerine. Rarity, even absence, of "pure" cases does not seem to me a conclusively adequate reason for abandoning useful clinical descriptive terms; especially since no one can deny the occurrence of difficulties in speaking, reading, or writing, and Dr. Head's own cases, needless to say, exemplify these admirably. If the absence of *separate* implication of any constituent in speech is to be advanced as an argument for the relinquishing of the above terms, exactly the same argument would apply to Dr. Head's own new classification of the varieties of aphasia into "verbal," "nominal," "syntactical," and "semantic" groups, since he himself admits that in most cases "two or more of these aspects of symbolic formulation are affected," and, again, "these terms are not intended to define the limits of the disturbance." In other words, since "pure" cases of these varieties never occur either, the argument for the abandonment of agraphic or alexic varieties on the ground of their never occurring in a "pure" state falls to the ground.

(3) If, now, we take Dr. Head's new subdivisions, we learn from him that in verbal aphasia the essential defect is one of word-formation; "words are evoked with difficulty and the vocabulary is greatly restricted; enunciation is slow and halting." In addition, writing may

be extremely difficult or almost impossible, and in any case "tends to show the same errors as articulatory speech." In nominal aphasia the patient has difficulty in naming letters, words, or objects. In addition, however, "writing is gravely affected," and the power of reading is greatly hampered. The third group, syntactical aphasia, is one where the patient talks jargon. Here also, however, "patients suffering from the more severe degrees of this affection cannot write a letter," and we are told that the disorder "is essentially one of auditory balance and rhythm." Finally, in semantic aphasia there is a want of recognition of the full significance of words and phrases. The patient "fails to comprehend the final aim or goal of an action"; he cannot "formulate symbolically a general conception."

Of the clinical occurrence of these types there is, of course, no question, and we owe a debt of gratitude to Dr. Head for the emphasis he has laid on them. Their content and significance have never been so closely examined and so admirably illustrated. In particular, it appears to me sufficient attention has never been paid in this country to the grammatical aspect of aphasia, and as I personally have long been aware of its importance and have much material bearing on it, I should like to take this chance of giving my support to Dr. Head's conclusions as far as this part of the subject is concerned.

At the same time, I do not see that the "new" types, so-called, in any way *run counter to* the hitherto accepted and familiar subdivisions. Verbal aphasia closely resembles ordinary motor aphasia; the nominal variety is one commonly observed, and I may say incidentally that it is in my opinion capable of sufficiently close localization. The neurologist is aware of the association between Mills' naming centre as it has been called, and integrity of function of the upper temporal lobe, and I have seen more than one excellent example of this defect in naming from strictly limited lesions in that part of the brain. Dr. Head includes much more than naming defects, however, in his nominal aphasia, as we have seen, though this naming defect is the special clinical feature. Syntactical aphasia, again, with its jargon-aphasia, has been described often enough in the group of sensory aphasia, and I showed a perfect example of this variety at a clinical meeting of the old Neurological Society. It will be remembered that, as already noted, Dr. Head agrees the disorder is essentially one of *auditory* "balance and rhythm."

The fourth classification leads me to what I think the most significant part of the new doctrine. Perusal of Dr. Head's illustration of semantic aphasia shows that it is largely identical with agnosia, in particular with that described by Liepmann and others as ideational agnosia. The examples furnished make this quite clear. Similar illustrations were given by myself from personally observed cases in my paper on apraxia.

Taking aphasic symptomatology as a whole, it appears to me that what Dr. Head has done is to make a new division which may be said in some ways to run across the classical divisions. Thus instead of speaking of agraphia and alexia, he speaks of verbal, nominal, and syntactical aphasia, but he admits that what is called agraphia or dysgraphia is a feature of all three, while alexia occurs at least in the second, if not in others. This again brings out the point to which I have referred above, that there is not, indeed there cannot be, any *subversion* of previous clinical knowledge in Dr. Head's work.

Dr. Head has not, as far as I have observed, used either of the terms agnosia and apraxia in his papers, obvious though it is that he is frequently describing apraxic or agnostic phenomena. Hence the significance of the remark already quoted from his paper, that in aphasia the disturbance extends beyond speaking, reading, and writing, and affects other mental activities. I believe the subject can best be understood if disorders of these "faculties," and the other mental activities mentioned by him, are looked at from the point of view of apraxia and agnosia. I do not wish to take up the time of the meeting further, except to sketch in a word what line I follow. I accept the general division of aphasia into receptive and executive defects; I hold that ordinary motor aphasia is but a part of motor apraxia, ordinarily called apraxia, and sensory aphasia of agnosia. The patient with auditory agnosia hears sounds, but does not recognize them, i.e., does not know their meaning. Among these sounds may be word-sounds, which he hears, but the content, the symbolic meaning, is for him unknown. The same is true of alexia as a variety of visual agnosia. Defects of this sort may certainly occur in unilateral lesions of the left hemisphere. On the executive side motor aphasia is a part of apraxia; the patient is not paralysed, but he cannot say what he wants to say, as the apraxic cannot do what he wants to do.

The analysis may be carried closer, and the clinical varieties of aphasia may be shown to have their counterpart on both executive and receptive sides in the matter of the "faculty" or "psychical activity" of music, and in the reckoning faculty. My view is that on the clinical side, which is all that for the moment concerns us, speech disorders, just as reckoning and music disorders, are parts of the wider impairments of function known as apraxia and agnosia.

the localization of the various functions had always been assumed to be too microscopical. Thinking out the matter on purely psychological grounds, knowing something of the localization of the motor function of the brain, and adopting Hughlings Jackson's views as to the representation of particular areas, he could not believe that such an important function as that of speech could be localized in any small area of the brain: it must require a vast representation. He believed that the area of the left side of the brain devoted to speech stretched from the foot of the third frontal convolution in front to the angular gyrus behind; and from the lower limits of the supramarginal convolution above to the middle and probably the inferior temporo-sphenoidal convolution below. This large area, including most of the island of Reil, he believed to be mainly devoted to speech (both internal and external) and intellect. He believed there were no sharply-divided compartments in this area where it could be said that this or that part of the cortex was solely devoted to particular elements of the speech function, but he did believe that in the main the hinder end of this area was mainly one in which visual impressions were received and elaborated into such a form that they were ready to be received in consciousness and interpreted as speech; and that in the same way the temporal convolutions were mainly devoted to re-representing auditory impressions as speech; whilst the frontal end was chiefly concerned with the emission of speech in the form of words spoken and written.

Dr. Head had told of a variety of lesions and a variety of results. There was a suggestion—nothing more—that whether there would be one type of aphasia or the other would be determined according to the intensity of the lesion, and not according to its localization. In this matter his experience was similar to that of the last speaker and others who had spoken that night. He thought there could be no doubt that a lesion of the hinder end of the speech area would give different results from those of a lesion in the fore end. But he thought that in all these cases there would be a depression of intellect, that the whole intellectual faculty would suffer to some extent in whatsoever portion the speech area was damaged. And he agreed with Dr. Collier that the more sharply defined was the type of aphasia, the more certainly would there be a subcortical as well as a cortical lesion. He did not think there was any sharp line of definition between motor and sensory cortex, but that they were intimately intermixed, and that as one passed to the fore end of the brain it became more motor, and as the occipital end was approached it became more sensual (visual), while on reaching the temporal lobe it was mainly auditory in type. He did not doubt that Dr. Head had explained his own cases, in the main, correctly. But, there again, he agreed with Dr. Wilson that those cases were not, essentially, any better for the purpose of determining localization than were the types members had seen. He had seen cases of bullet wounds in which the brain had been exposed by operation, cases in which the missile had crushed through the dura and those which had suffered concussion effects through a slanting blow, and it had been difficult to define the limits of the lesion, either in the surface direction or in depth. He thought Dr. Head would agree that, though it was clear that the lesions in his cases were mainly cortical, yet, to a considerable but varying extent, they might be subcortical.

He did not yet know exactly what Dr. Head meant in regard to the type of theory of which Sir James Purves Stewart spoke. He (the speaker) thought the main question at issue as between neurologists to-day was as to whether there was an intermediate cortical "centre" through which impulses passed, whether they were elaborated as coming from the sensory side and were coded as they were passing out in the emissary form. He agreed that there was, as yet, no absolute proof that the visual impulses did not go directly into the main mass of the speech area, that they were entering into intelligence in such a form that one could say that at such a particular place they had been re-represented or integrated to a higher stage, so that they could then reach consciousness. But he felt very little doubt that the process of integration was going on; he thought it was operative in an area not sharply defined from the remainder of the speech area, and that neurologists could use such a localization as had been worked out in the past very valuably from the point of view of localization of speech function.

J. HERBERT PARSONS, F.R.C.S.[1]: In his two admirable lectures on aphasia Dr. Henry Head has not defined precisely the psychological aspects of the question. Doubtless these aspects were fundamental to his thesis and were ever present in his mind, though the exigencies of the circumstances prevented their exhaustive discussion. The fact that he has laid so much stress on "meaning," as shown by the introduction of the term "semantic aphasia," is ample evidence that the omission is not due to oversight; it proves, in fact, that accurate psychological principles are the very foundation of his inspiration. It is, however, regrettable that these aspects have not been treated more definitely; in the first place, because they afford the most valuable support to his views; and in the second place, because their omission is likely to lead to misconception. Indeed, there is reason to think that such misconception has already arisen. It is readily explicable, for neurologists most interested in the subject naturally view it from the anatomical and physiological standpoint, rather than from the psychological, owing to their training and clinical experience. They are therefore liable to ignore or misconceive the exact rationale of pathological anatomical lesions in the genesis of disorders of the highest psychic activities.

---

[1] These remarks on "The Psychology of 'Meaning' in its Relation to Aphasia" were handed in during the discussion but were not actually delivered.

The emphasis laid upon the semantic aspects of aphasia is the sign of a great advance in our conception of the disease, an advance inspired by the immortal work of Hughlings Jackson, confirmed by the previous researches of the distinguished author of the lectures, and founded upon the psychological aspect of the subject. Sound scientific psychology is therefore the crux of the question, and has hitherto not received the attention it deserves.

The psychology of "meaning" has been curiously neglected, and it is only of recent years, largely owing to G. F. Stout and the biological psychologists, of whom Lloyd Morgan is the outstanding example, that it has been analysed according to scientific principles. The work hitherto accomplished in this field is almost limited to the lower psychic activities, especially at the perceptional level. But "meaning" from the biological and psychological points of view has undergone profound phylogenetic and ontogenetic evolution, and speech is the symbolic manifestation of the highest and most complex form of "meaning."

At the lowest biological level, where the reflex arc constitutes the whole nervous mechanism, we can only conjecture the conscious accompaniments of nervous activity. It is probable that it is a vague undifferentiated sentiency, possessing a maximum of affective tone and a minimum of cognition. To it the term "meaning" is scarcely applicable, for the response is little more than a tropism: yet it would be unwise to deny the presence of a plus or a minus affective tone—pleasure or unpleasure—and this is the primitive germ of "meaning." In the higher species, in accordance with a very general psychological law, the conscious accompaniments of the reflex act have been suppressed.

At a slightly higher biological level, reflex arcs have become co-ordinated and integrated, and the response assumes the character of instinctive reaction. It is unnecessary to labour over the conscious accompaniments of the inceptive stage of the instinctive level, for it passes by imperceptible gradations into the perceptual level which is the summit which most lower mammals have attained.

At the perceptual level, in any given first experience, sensations are aroused in the organism by physical stimuli from the outer world. Psychologically, as an impression of the given situation, sensations are objective. They undergo differentiation and subjective selection. The selected items are then reintegrated. The reintegration is of the nature of a truly creative synthesis, and issues in the form of an entirely new order of consciousness. To resume in other words, the relatively undifferentiated psychoplasm—to use a useful, but awkward and too concrete term—is differentiated into specialized affective and cognitive elements, which are reintegrated, thus undergoing a synthesis which is the "meaning" of the given experience. Perceptual "meaning," suffused with affective tone, issues in instinctive conative activity. This itself by "backstroke" supplies new modifying stimuli to the already altered psychoplasm. For, in addition to the satisfaction or unsatisfaction of the successful or obstructed instinctive reaction, the motor activities are accompanied by somatic afferent impulses modifying the cœnæsthesis which forms an important part of the conscious state.

Thus, at the end of the completed reaction the "meaning" has become enriched and complicated on the affective side by the impact of the afferent impulses derived from somatic and visceral sources, and on the cognitive side by the results of the conative activity. This altered "meaning" is stored up, and, though depressed below the threshold of consciousness, is capable of being revived and recognized on the occurrence of a cognate experience.

On the next occasion of a similar experience, in addition to the factors of the first occasion are the factors derived from recognition and revival of this concrete event which has been lived through. Learning by experience is the utilization of these revived factors, and consists chiefly in the addition of cognitive elements. The assimilation, differentiation, subjective selection, integration and synthesis of the already more plastic psychoplasm results in a higher, more complex type of "meaning"—a "meaning" which we recognize as of higher grade and explain as an intelligent modification of the hitherto essentially instinctive reaction.

In the human organism the instinctive elements, as well as innate dispositions of a higher order, are already present in immature form as inherited characteristics. The simpler syntheses of the lower mammal are complicated by this wealth of innate factors. Not only is the first experience thus complicated, but each succeeding experience is further complicated, and experiences which are only remotely similar add their quota to the already complex mass of factors, inherited and acquired. Thus, even at the perceptual level "meaning" becomes extremely complex.

With the evolution of free ideas, and still more under the fully developed assimilation and apperception [illegible] conceptual thought, the complexity is further increased.

Hitherto no special stress has been laid upon the social environment of the individual, but we have now learnt that man is a "socius" from very early infancy. Long before a child can talk the "meanings" of the different phases of his experience of the outer world acquire characteristics which are imprinted upon them by the assimilation of special qualities derived from intercourse with his fellow creatures, of whom, of course, his mother is the most important. Though this assimilation should be regarded as a perpetual recurrence of the cycle of differentiation, subjective selection, integration, and creative synthesis, the ultimate results are equivalent to an interaction of old and new "meanings," resulting in an infinity of still newer, richer and more refined "meanings." At this stage conative activities themselves assume a synergy at a higher level, which is the dawn of true volition. The synthesis of the child's mental processes into a "self" is the product of his social intercourse, and his conative activities now show a projicience hitherto absent; to the outside observer his gestures are no longer merely passive signs of his mind's activities, but active indications of his feelings and desires. This is the dawn of language.

It would, of course, be a gross error to imagine that this brief sketch gives any true idea of the complexity of the evolution of "meaning." There is another aspect which is particularly illuminating from the point of view of aphasia, especially in association with Dr. Head's work. The analogy between the primitive undifferentiated instinct—with its "all or none" response, comparable to the "mass-reflex" of the isolated spinal cord—and protopathic sensation, has been pointed out and exhaustively elaborated by Dr. W. H. R. Rivers in his "Instinct and the Unconscious." Similarly the differentiation of the primitive instincts, with the partial suppression of the vague elements belonging to the protopathic level, is comparable to epicritic sensations.

These essentially biological and physiological aspects form the foundation of the more purely psychological aspects already considered, and are of the greatest importance in the elucidation of the phenomena of aphasia.

Enough has been said to show that "meaning" at the speech level is extraordinarily complex. Yet the methodology of analysis is now taking shape, and its application has already elucidated many of the obscurities at the perceptual level. Much yet remains to be done at higher levels. Even when this is done there yet remains the enormous field of the psychology of the concrete individual, a matter of no small importance in dealing with aphasic patients of different degrees and varieties of nature and nurture. The "meaning" of a given situation will differ for different individuals according to their inherited instincts and innate dispositions as well as their acquired experience, especially that derived from their social environment. These differences will manifest themselves in their motor responses, and above all in speech.

I have hitherto considered only the organization of thought, using that term in a wide sense. The difficult question how far the organization of conceptual thought is dependent upon speech—"internal speech," in Dr. Head's terminology—has recently received considerable attention (*British Journal of Psychology*, General Section, October, 1920, pp. 55 to 104). Certain it is that some re-synthesis occurs in the symbolization of thought which is the essential feature of speech, and that a further re-synthesis occurs in the transformation of internal into external speech. Light is thrown upon this part of the process by the study of the development of speech in the child and particularly by the psychology of reading. One elementary fact is demonstrated by the latter study, viz., that the grouping of concepts gradually develops a wider span—from the reading of letters, or at any rate monosyllabic groups of letters, the child progresses to the apprehension of groups of words. I have dealt with this portion of the subject elsewhere (*British Medical Journal*, August 22, 1914).

When it is realized that the neurologist attacks the problem at the most complex stage of its development, and that he is confronted with focal anatomical lesions which are difficult to reconcile with the psychological complexities, it is not surprising that his explanations should be crude and of only partial validity. Much of the crudity is due to the unfortunate prevalence of the term "centre." So long ago as 1904, speaking of various pupillary "centres" which have been described, I wrote: "One is inclined to wish that this term might be abolished from neurology, so great is the ambiguity and confusion to which it has given rise. The intercommunications between the cells of the central nervous system are so complex that there are almost innumerable alternative paths whereby any given group of efferent cells may receive excitatory impulses. Our aim should be, first to determine the efferent cells governing any given movement, second to determine as far as is possible which are the afferent tracts which can lead directly or indirectly to those cells, and third to find out *how* each of those afferent tracts leads to the efferent cells. The last problem is the most difficult, and we shall find that many of the afferent tracts are very devious, and have many cell-stations on their way. Each of these may be dignified with the title 'centre,' or better, none of them" ("The Neurology of Vision," 1904, p. 50). The term—and the train of thought associated with it—is eminently unsuitable when dealing with the higher psychic phenomena, such as "meaning" and the speech which subserves it. Scientifically, the unity of the mind is correlated with a synthesis of nervous activities which are so widespread throughout the nervous system that the term "centre" is ludicrously inadequate. Yet these syntheses are dependent for their perfection upon congeries of factors which are in some degree accessible to analysis. In disorders of speech for instance, lesions on the afferent side, of which the auditory are of prime importance, will cut off some of the pabulum for differentiation, subjective selection, integration and synthesis. Such cases have been described clinically as sensory aphasias.

Dr. Head has somewhat forcibly deprecated the use of such terms as motor and sensory aphasia, alexia, agraphia, anarthria, and so on, but his protest belongs to the order of counsels of perfection. That such terms have often been unwisely chosen and inaccurately applied may well be granted, but their use as descriptive terms can scarcely be avoided with any great degree of consistency. In studying a complex "higher level" subject, of which the fundamentals are very imperfectly known, the lumping together of allied factors under one name is not only convenient but unavoidable if awkward circumlocutions are to be avoided. Doubtless semantic aphasia is a form of agnosia: both terms have their uses as well as their abuses. It is a matter of precise definition and consistent adhesion to the defined significance.

The study of aphasia is such a pre-eminent example of the necessity for the collaboration of psychologists and neurologists and of the advantages which would accrue to both sciences by more intimate co-ordination that it is, I hope, a sufficient excuse for one so unversed in the clinical aspects of the subject having the temerity to make this communication.

Dr. HEAD (in reply) said he could not imagine a task more difficult than to be questioned by the various speakers and to have to answer them unprepared. He understood Dr. Collier to be in general agreement with the clinical aspect of what he (Dr. Head) had put forward. He could not understand why Dr. Collier said that in spite of the fact that the terms used were fundamentally incorrect, and "physiologically, anatomically, and psychologically impossible," he intended to keep to them. It was for such reasons he (the speaker) had been bold enough to try to replace them by something which would be more fundamentally correct, more physiologically adequate, more psychologically possible. In speech they were dealing with a high-grade psychical aptitude far above the level of sensation. This function demanded the integration of a number of other functions before it could be carried out, and in this way speech seemed to resemble sensation; either could be upset by lesions of the brain, which did not necessarily destroy the whole act, but destroyed it in part. The mere fact that speech could be disturbed in part showed that those areas of the cortex which were attacked by these various lesions were not all of equal value. He gathered from Dr. Collier that he considered most of the lesions which produced these disturbances affected either the entering or the emissary path—i.e., in the pure forms; but with lesions of the entering path they were not concerned with speech at all, but with a disorder of some aspect of sensation. On the emissive side such a lesion should produce a pure anarthria.

Proceeding to the discussion of syntactical aphasia, he would try to unite the two parts of Dr. Collier's statement together. In the first part of his discussion on syntactical aphasia, Dr. Collier had hit on the explanation he (the speaker) had given of its origin. If a man had taken too much alcohol his legs became ataxic; he struck against chairs and tables in his progression, and the repercussion on himself told him he had not proper control of his legs. But if at the same time his speech was disorderly, he did not learn this through his somatic sensations, he knew it because he heard his speech to be wrong. Syntactical loss was due to the fact that the man could speak, but had lost regulating power over the rhythm and balance of what he said. The actual words, like all words spoken in these disturbances of language, were representative of the parts not affected. He was glad to find that, in this matter, Dr. Collier and he had hit upon the same explanation.

He was sorry to see that, later on, Dr. Collier used the terms "visual speech" and "auditory speech," as he (Dr. Head) did not know what they were; there was speech, but not visual speech or auditory speech. When a person was speaking, his actions were carried out by physiological processes above the visual and the auditory levels. In most cases of aphasia, image-formation was not definitely impaired. True, there might be a combination of loss of image-formation and loss of speech. One of the best cases on record was that which Stauffenberg worked out in v. Monakow's laboratory in 1913. This was certainly a case of aphasia combined with what Hughlings Jackson would have called imperception.

He would like to correct the statement that his patients were selected because the lesion was thought to be cortical; with these wounds no one could be certain whether a lesion was cortical or subcortical. His object had been to see what were the actual forms assumed by destruction of speech—i.e., a high psychical function—in consequence of various organic lesions. It was impossible to suppose that the relation between organic destruction and the loss of psychical function stood in the relation of so much loss of cortex to so much disorder of psychical function. The effect of an organic lesion of the brain on the functions of speech might be compared with the result on an electric light installation of destroying a varying number of the accumulators; the area of the brain supplied the vital force, which was the basis of the physiological energy which underlay this complicated psychical act. It might easily happen that it did not disintegrate, but rather lowered the general activity and vitality of the power of speech. As a fact, however, injury to one portion of the brain was more likely, under favourable conditions, to lead to the breaking up of speech in certain directions. But before attempting to decide the situation of the lesion which produced some specific disorder of speech, it was absolutely necessary to ascertain what the disturbance was. It was of no use trying to localize the position of an unknown function on the surface of the brain. That was why he had kept away from all questions of localization at present; he wanted first to determine what function was disturbed, before putting forward views as to what lesion was likely to produce it.

Most of the speakers had accused him of being an empiric. He was delighted to hear it. The whole point in his papers was that he wanted to follow the line of English empiric philosophy dating from Locke and passing into medicine through Hughlings Jackson. The latter said, in a famous passage, "Put down what the patient does get at, and avoid all such terms as amnesia, &c."

The next point on which he wanted to insist was that these functions were not faculties; one speaker used the word "faculty" three or four times. "Faculties" came from mediæval philosophy, and had now, fortunately, been got rid of in psychology. The use of such words as "general understanding," "aphasia," "amnesia," "memory" had been deprecated by Hughlings Jackson, who pointed out that there was no such thing as memory apart from things remembered, and no consciousness apart from conscious actions. The only possible way to find out what happened in consequence of an organic lesion was, not to use words such as "reading" and "writing" which were only *a priori* conceptual terms, but to observe what did actually happen. Had he been bold enough, he would have liked to have spoken of the "*x* group of functions," which broke up into the "*a*, *b*, *c* and *d* groups" But he dare not do that He had been obliged to give them names, and it was those names which were going to lead him to perdition; because everyone would settle upon them and think they all meant something he did not intend; another set of conceptual terms would have been invented, to load up this unfortunate subject and obscure it further. There were no such things as "types" of aphasia. A vast group of functions were employed in the acts called speaking, reading and writing, and it was these which split up in certain ways to produce the clinical forms of disordered speech.

Sir James Purves Stewart simplified the question by dividing speech affections into psycho-motor or apraxia, and psycho-sensory or agnosia. It seemed to him (the speaker) that this was a matter of words; he did not know what psycho-motor meant, and apraxia was only a convenient term for certain aspects of defective cerebral activity. Neither did he know what "psycho-sensory" meant; the sensory side of speech must consist of sensations. When it was said that the psycho-sensory aspect of speech was agnosia, this was only saying in Greek that the patient did not appreciate the significance of impressions which came to him from without. With regard to the cases described, it was extraordinary how anybody, working under the conditions surrounding Sir James Purves Stewart in Gallipoli and elsewhere, should have been able to take such photographs and such elaborate notes It filled him with admiration But all these cases tended to have their value decreased because they were seen in the acute period, a period which in many cases extends over many months. It was difficult to say what specific form would be assumed afterwards by cases showing such a widespread lowering of vitality. Cases 2 and 3 appeared to have had verbal loss, with difficulty in spelling. If they could have been watched over a long enough period, he thought they would be found to be verbal aphasics. No. 4 seemed to be too diffuse to be at all definite, but No. 5 was a severe example of nominal aphasia.

Dr. Wilson seemed to think that aphasia and apraxia could be put together under one heading. Both were convenient words if they were not carried to definition. The moment one drove "apraxia" to definition one saw it did not exist. There were forms of apraxic action, which no one had described better than had Dr. Wilson himself; but when one used the word "apraxia" to explain loss of speech, it did not seem to explain more than the most simple of aphasias. He wished to get rid of all such terms.

Dr. Stanley Barnes stated that the impression had got about that localization had gone by the board. If by this was meant the localization of "alexia," "agraphia" and "aphasia," he was glad he had given the correct impression. By localization he understood that affections of certain parts of the brain were liable to produce breakdown of this group of functions in one direction rather than in another. The anatomical basis of that form of localization he hoped to put before the Section on a future occasion. He did not believe in localization as it was ordinarily stated; he did not believe in the localization of what was called "motor aphasia," or "sensory aphasia," because he did not believe in the existence of motor and sensory aphasia.

With regard to the cortical or subcortical nature of the destruction, no one would suppose that gunshot wounds were other than gross lesions, and the wonder was that after the shock had passed off any of them were found to have produced specific loss of function. Dr. Barnes laid stress on a general intellectual defect; but psychologists to-day did not believe in general intellectual defects. This could be seen in the case of memory. Jackson said there was no such thing as general memory; there were only definite things remembered. Speech was a form of intellectual activity; therefore in saying there was a defect of those processes which were responsible for speech, it was assumed there was a defect of intellect. But this was a specific loss of function and there was not that gross widespread defect of intellectual aptitudes as was found in the slighest case of dementia.

PUBLICATIONS RECENTLY RECEIVED.

Studies in Word Association. Under the direction of C. G. JUNG, M.D., LL.D. Authorized translation by Dr. M. D. EDER. Pp. 575. London: William Heinemann, 1918.

Nearly half of this large volume is devoted to the study of the value of the response to the stimulus word. Jung and his co-workers have for this purpose investigated thirty-eight normal persons in great detail under varying conditions, and they have brought out interesting and in some ways unexpected results. As might have been surmised, they were able to show that on distraction whether external or internal, and in states of fatigue, the associations were of a more superficial kind than when these factors were absent; but a surprising result, obtained from the study of normal people, consisted in the fact that, other things being equal, educated people gave more superficial reactions than the uneducated. "The uneducated knows the word less as a verbal sign than as something with a meaning. Hence, single words are apprehended by them according to their meaning in some imaginary sentence; to the educated the stimulus word remains merely a word without any special meaning." From the different kinds of reaction which the experimenters classified, Jung has been able to make further progress with his psychological types.

To the psycho-analyst the main outcome of these investigations is that the importance of marked prolongation of reaction time as a complex indicator remains unimpaired. Though fatigue and external distraction may cause prolongation, they do not do so with the same success nor in the same selective way as the internal distraction of emotion.

In an elementary chapter Professor Bleuler seeks to demonstrate the fact of the unconscious. Unfortunately nearly all his proofs are for something of the nature of the fore-conscious or of a co-conscious which reasons after the same fashion as the conscious, the part of the subconscious which enables us by mental process to avoid jostling passengers in the street when we are thinking of something else. That is not a part of mental process which is in need of much demonstration. Its conception arouses no opposition, and it is probably accepted by everyone. What is needed is clear proof—and such is not difficult to find—of a subconscious whose contents never emerge into consciousness except by distortion, although they have great influence on our thoughts and actions.

There are useful chapters on word reactions in psycho-analysis and treatment, on the psycho-galvanic reaction and on associated physical changes.

It may be said that the work is one of great value, that it strengthens in every way the doctrines of psycho-analysis; for it is a record of facts derived from painstaking work, and is free from the speculative views which run through much of Jung's later work.

Dr. Eder deserves the highest praise for the translation; so excellent is it that the book might have been originally written in English.

The Psychology of the Special Senses and their Functional Disorders. (The Croonian Lectures delivered before the Royal College of Physicians in June, 1920.) By ARTHUR F. HURST, M.A., M.D., F.R.C.P. Pp. 123. London: Oxford University Press, 1920.

In these lectures Dr. Hurst gives an account of his observations on cutaneous sensibility, visceral pain, superficial reflexes and affections of the special senses, particularly in connection with the war neuroses. He accepts Babinski's definition of hysteria as "a condition in which symptoms are present which have resulted from suggestion and are curable by psychotherapy." He also agrees that all the ordinary forms of loss of somatic sensibility and limitation of the visual fields found in this disease are suggested to the patient; he gives instances to show, however, that in some cases during the war the suggestion came from a gross nerve injury. But in support of this view, with which all will agree who have had the opportunity of making observations on wounded soldiers, he cites cases where the "anæsthesia" occupied the whole area usually given by anatomists as the supply of the ulnar and median nerves. There is no note to show how this loss of sensibility was discovered. The usual form assumed in our experience by such hysterical conditions is for the whole anatomical area attributed to the injured nerve to be insensitive to prick, a condition which never occurs even after complete division of either the ulnar or median nerves. It is not an hysterical perpetuation of the condition produced by the injury but a superposed state due to suggestion by the medical examiner.

The author believes that the abdominal and plantar reflexes can be abolished in uncomplicated hysteria, although precautions are taken to protect the skin of the abdomen and the foot from cooling. He also considers that in the larger number of instances tenderness of the skin in cases of visceral disease is due to suggestions on the part of the observer.

He gives several cases of hysterical deafness and states that the auditory motor reflex may be absent and vestibular tests alone can show whether this condition is hysterical or not. Total blindness, even when accompanied by loss of reaction of the pupils to light, may be hysterical and undergo rapid cure.